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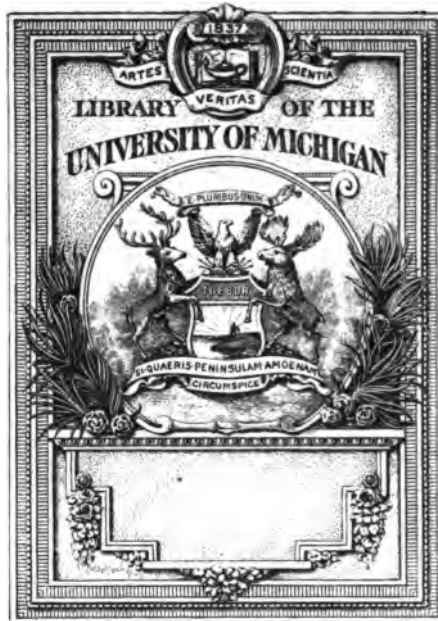
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MEDICO-CHIRURGICAL TRANSACTIONS.

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LONDON.

VOLUME THE SIXTY-SECOND.

LONDON:
LONGMANS, GREEN, READER, AND DYER,
PATERNOSTER ROW.

1879.

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1879.

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- 1821. JOHN COOKE, M.D.
- 1823. JOHN ABERNETHY.
- 1825. GEORGE BIRKBECK, M.D.
- 1827. BENJAMIN TRAVERS.
- 1829. PETER MARK ROGET, M.D.
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- 1833. JOHN ELLIOTSON, M.D.
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- 1851. JOSEPH HODGSON.
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- 1855. CÆSAR HENRY HAWKINS.
- 1857. SIR CHARLES LOCOCK, BART., M.D.
- 1859. FREDERIC CARPENTER SKEY.
- 1861. BENJAMIN GUY BABINGTON, M.D.
- 1863. RICHARD PARTRIDGE.
- 1865. SIR JAMES ALDERSON, M.D., D.C.L.
- 1867. SAMUEL SOLLY.
- 1869. SIR GEORGE BURROWS, BART., M.D., D.C.L.
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The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Sci. Com.* is attached to the names of those who have served on the Scientific Committees of the Society.

OCTOBER, 1879.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

- 1846 *ABERCROMBIE, JOHN, M.D., Physician to the Cheltenham General Hospital ; 13, Suffolk square, Cheltenham.
- 1877 ABERCROMBIE, JOHN, M.B., Hospital for Sick Children ; 49, Great Ormond street.
- 1851 *ACLAND, HENRY WENTWORTH, M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales ; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.
- 1847 ACOSTA, ELISHA, M.D., 24, Rue de Luxembourg, St. Honoré, Paris.

Elected

- 1852 ADAMS, WILLIAM, Surgeon to the Great Northern Hospital and to the National Hospital for Paralyse and Epileptic; Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.
- 1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde park.
- 1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.
- 1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.
- 1866 ALLBUTT, THOMAS CLIFFORD, M.A. and M.D., F.L.S., Lecturer on the Practice of Physic at the Leeds School of Medicine, and Physician to the Leeds General Infirmary; 35, Park square, Leeds. *Trans.* 3.
- 1863 ALTHAUS, JULIUS, M.D., Physician to the Infirmary for Epilepsy and Paralysis; 36, Bryanston street, Portman square. *Trans.* 2.
- 1878 AMPHLETT, EDWARD, M.B., Assistant Surgeon to Charing Cross Hospital; 40, Weymouth street, Portland place.
- 1862 ANDREW, EDWYN, M.D., Hardwick House, St. John's Hill, Shrewsbury.
- 1862 ANDREW, JAMES, M.D., *Secretary*, Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square. S. 1878-9. *Trans.* 1.
- 1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.
- 1878 ARNOLD, JOHN, Medical Officer of Health; Trinidad.
- 1870 ARNOTT, Rev. HENRY, Braeside, Beckenham.
- 1819 †ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire, and 36, Sussex gardens, Hyde park. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Lib. Com. Trans.* 8.
- 1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.

Elected

- 1836 BAIRD, ANDREW WOOD, M.D., Physician to the Dover Hospital; 7, Camden crescent, Dover, Kent.
- 1851 *BAKER, ALFRED, Surgeon to the Birmingham General Hospital; 20A, Temple row, Birmingham.
- 1873 *BAKER, J. WRIGHT, Senior Surgeon to, the Derbyshire General Infirmary; 102, Friargate, Derby.
- 1865 BAKER, WILLIAM MORRANT, Assistant Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; Surgeon to the Evelina Hospital for Sick Children; 26, Wimpole street, Cavendish square. C. 1878-9. *Lib. Com. Trans.* 5.
- 1869 BAKEWELL, ROBERT HALL, M.D., Ross, Westland, New Zealand.
- 1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Surgeon General; Coombe Lodge, Wimbledon Park. C. 1852-3. V.P. 1860-1. T. 1872. *Lib. Com. Trans.* 2.
- 1848 †BALLARD, EDWARD, M.D., Inspector, Medical Department, Local Government Board; 12, Highbury terrace, Islington. C. 1872. V.P. 1875-6. *Lib. Com. Trans.* 5.
- 1866 *BANKS, JOHN THOMAS, M.D., Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to the Coombe Hospital; 10, Merrion square east, Dublin.
- 1847 †BARCLAY, ANDREW WHYTE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; Medical Officer of Health for Chelsea; 23A, Bruton street, Berkeley square. S. 1857-60. L. 1861-2. C. 1865-6. V.P. 1872-3. *Trans.* 2.
- 1879 BARKER, ARTHUR EDWARD JAMES, Assistant Surgeon to, and Assistant Professor of Clinical Surgery at, University College Hospital; 28, Welbeck street, Cavendish square.
- 1862 BARKER, EDGAR, 21, Hyde park street.
- 1833 †BARKEE, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2. *Trans.* 6.

Elected

- 1876 BARLOW, THOMAS, M.D. and B.S. Lond., Assistant Physician to the London Hospital, and to the Hospital for Sick Children, Great Ormond Street; 10, Montague street, Russell square.
- 1861 BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; 15, Harley street, Cavendish square. C. 1877-8. *Lib. Com. Trans.* 4.
- 1864 BARRATT, JOSEPH GILLMAN, M.D., 8, Cleveland gardens, Baywater.
- 1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.
- 1859 BARWELL, RICHARD, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; 32, George street, Hanover square. C. 1876-77. *Trans.* 7.
- 1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; 20, Queen Anne street, Cavendish square. *Trans.* 1.
- 1874 BAXTER, EVAN BUCHANAN, M.D., Professor of Materia Medica at King's College, London; Assistant Physician to King's College Hospital; Examiner in Materia Medica at the University of London; 28, Weymouth street, Portland place.
- 1875 BEACH, FLETCHER, M.B., Medical Superintendent, Metropolitan District Asylum, Darenth, near Dartford, Kent.
- 1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Trans.* 1.
- 1860 *BEALEY, ADAM, M.D., M.A. Camb., Oak Lea, Harrogate.
- 1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.
- 1871 BECK, MARCUS, M.S., Assistant Surgeon to University College Hospital; 30, Wimpole street, Cavendish square.

Elected

- 1858 BEGLEY, WILLIAM CHAPMAN, A.M., M.D., late of the Middlesex County Lunatic Asylum, Hanwell; 26, Saint Peter's square, Hammersmith. C. 1877-8.
- 1819 †BELL, THOMAS, F.R.S., F.L.S., The Wakes, Selborne, Hants. C. 1832-3. V.P. 1854. *Trans.* 1.
- 1871 BELLAMY, EDWARD, Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Professor of Anatomy in the Science and Art Department, South Kensington; 17, Wimpole street, Cavendish square. *Lib. Com.*
- 1847 BENNET, JAMES HENRY, M.D., The Ferns, Weybridge, and Mentone.
- 1877 BENNETT, WILLIAM HENRY, Surgeon to the Belgrave Hospital for Children; 5, Savile row, Burlington gardens.
- 1845 †BERRY, EDWARD UNWIN, 76, Gower street, Bedford square.
- 1820 BERTIN, STEPHEN, Paris.
- 1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 63, St. Giles's street, Norwich.
- 1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.
- 1815 †BILLING, ARCHIBALD, M.D., F.R.S., Member of the Senate of the University of London; 34, Park lane. C. 1825. V.P. 1828-9.
- 1878 BINDON, WILLIAM JOHN VEREKER, M.D., 2, Elm Villas, Kilburn.
- 1854 BIRD, PETER HINCKES, F.L.S., 1, Norfolk square, Sussex gardens, Hyde park.
- 1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.
- 1849 BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6.

Elected

- 1851 †BIRKETT, JOHN, F.L.S., *Vice-President*, Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 59, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879. *Sci. Com.* 1. *Lib. Com. Trans.* 8.
- 1866 BISHOP, EDWARD, M.D., Cintra park, Upper Norwood.
- 1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.
- 1865 BLANDFORD, GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 71, Grosvenor street.
- 1867 BLOXAM, JOHN ASTLEY, Assistant-Surgeon to, and Teacher of Operative Surgery in, Charing Cross Hospital; Surgeon for Out-Patients to the Lock Hospital; Junior Surgeon to the West London Hospital; 8, George street, Hanover square.
- 1823 BOJANUS, LOUIS HENRY, M.D., Wilna.
- 1846 †BOSTOCK, JOHN ASHTON, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 73, Onslow gardens, Brompton. C. 1861-2. V.P. 1870-71. *Sci. Com.* 1.
- 1869 BOURNE, WALTER, M.D. [care of the National Bank of India, 80, King William street, City.]
- 1870 *BOWLES, ROBERT LEAMON, M.D., 8, West terrace, Folkestone.
- 1841 †BOWMAN, WILLIAM, F.R.S., F.L.S., Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Lib. Com. Trans.* 3.
- 1862 BRACE, WILLIAM HENRY, M.D., 7, Queen's Gate terrace, Kensington.

Elected

- 1874 BRADSHAW, A. F., Surgeon-Major; Surgeon to the Rt. Hon. the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]
- 1867 *BRETT, ALFRED T., M.D., Watford, Herts.
- 1876 BRIDGES, ROBERT, M.B., Casualty Physician to St. Bartholomew's Hospital; Assistant Physician to the Hospital for Sick Children; and Physician to the Great Northern Hospital; 52, Bedford square.
- 1867 BRIDGEWATER, THOMAS, M.B. Lond., Harrow-on-the-Hill, Middlesex.
- 1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Physician to the London Fever Hospital; 34, Seymour street, Portman square. *Trans.* 3.
- 1851 †BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Lib. Com. Trans.* 2. *Pro.* 1.
- 1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.
- 1860 BROWN-SÉQUARD, CHARLES EDOUARD, M.D., F.R.S., Laureate of the Academy of Sciences of Paris; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Paris. *Sci. Com.* 1.
- 1878 BROWNE, JAMES CRICHTON, M.D., 7, Cumberland Terrace, Regent's Park.
- 1874 BRUCE, JOHN MITCHELL, M.D., Assistant Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 60, Queen Anne street. *Trans.* 1.
- 1871 BRUNTON, THOMAS LAUDEB, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica at, St. Bartholomew's Hospital; 50, Welbeck street, Cavendish square.

Elected

- 1860 BRYANT, THOMAS, Surgeon to, and Lecturer on Surgery, at Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873-4. *Sci. Com.* 1. *Lib. Com. Trans.* 8. *Pro.* 1.
- 1855 BRYANT, WALTER JOHN, M.R.C.P. Edinb.; Physician to the Home for Incurable Children, Maida vale; 23A, Sussex square, Hyde park gardens.
- 1823 BUCHANAN, B. BARTLET, M.D.
- 1864 BUCHANAN, GEORGE, M.D., Inspector, Medical Department, Local Government Board; 24, Nottingham place, Marylebone road.
- 1864 BUCKLE, FLEETWOOD, M.D.
- 1876 BUCKNILL, JOHN CHARLES, M.D., F.R.S.; 39, Wimpole street, Cavendish square.
- 1839 BUDD, GEORGE, M.D., F.R.S., Consulting Physician to the Seamen's Hospital, Greenwich; Ashleigh, Barnstable. C. 1846-7. V.P. 1857. *Trans.* 5.
- 1833 †BURROWS, SIR GEORGE, Bart., M.D., D.C.L., F.R.S., Physician in Ordinary to H.M. the Queen; Consulting Physician to St. Bartholomew's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V. P. 1849-50. P. 1869-70. *Lib. Com. Trans.* 2.
1837. †BUSK, GEORGE, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital, Greenwich; Member of the Senate of the University of London; 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Lib. Com. Trans.* 4.
- 1873 BUTLIN, HENRY TRENTHAM, Demonstrator of Practical Surgery to St. Bartholomew's Hospital; Assistant Surgeon to the West London Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 3.
- 1871 BUTT, WILLIAM F., 25, Park street, Park lane.
- 1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square.

Elected

- 1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.
- 1861 CALLENDER, GEORGE WILLIAM, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to the Charter House; 7, Queen Anne street, Cavendish square. C. 1874. *Trans.* 4. *Sci. Com.* 1.
- 1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde park.
- 1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 69, Wimpole street, Cavendish square, W. *Trans.* 1.
- 1845 †CARTWRIGHT, SAMUEL, late Professor of Dental Surgery at King's College, London, and Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.* 1.
- 1879 CARTWRIGHT, S. HAMILTON, Professor of Dental Surgery at King's College; 32, Old Burlington street.
- 1868 CAVAFY, JOHN, M.D., Senior Assistant-Physician to, and Lecturer on Physiology at, St. George's Hospital; 2, Upper Berkeley street, Portman square.
- 1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 58, Welbeck street, Cavendish square.
- 1845 †CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.
- 1844 †CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital; Consulting Physician to the Lock Hospital; Shrubs Hill House, Sunningdale, Staines. C. 1861. V.P. 1867. L. 1869-72. *Lib. Com. Trans.* 1.
- 1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

Elected

- 1849 CHAPMAN, FREDERICK, Old Friars, Richmond Green, Surrey.
- 1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; 10, Mitre court Chambers, Temple.
- 1868 CHEADLE, WALTER BUTLER, M.D., Assistant-Physician to, and Lecturer on Pathology and on Skin Diseases at, St. Mary's Hospital; Assistant Physician to the Hospital for Sick Children; 2, Hyde park place, Cumberland gate.
- 1873 *CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.
- 1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital, and to the Margaret Street Infirmary for Consumption; 63, Grosvenor street, Grosvenor square.
- 1872 CHRISTIE, THOMAS BEITH, M.D., Medical Superintendent, Royal India Asylum, Ealing.
- 1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square.
- 1860 CLARK, ANDREW, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 16, Cavendish square. C. 1875.
- 1879 CLARK, ANDREW, Assistant Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 19, Cavendish place, Cavendish square, W.
- 1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; 63, Warrior square, St. Leonards-on-Sea. S. 1847-9. V.P. 1855-6. *Lib. Com. Trans.* 5.
- 1848 †CLARKE, JOHN, M.D., 42, Hertford street, May Fair. C. 1866.
- 1866 CLARKE, WILLIAM FAIRLIE, M.D., M.A. Oxon., Southborough, Tunbridge Wells. *Trans.* 2.

Elected

- 1842 †CLAYTON, OSCAR MOORE PASSEY, Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.
- 1853 CLOVER, JOSEPH THOMAS, 3, Cavendish place, Cavendish square. C. 1873.
- 1857 COATES, CHARLES, M.D., Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.
- 1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free Hospital; 4, Suffolk place, Pall Mall East. *Trans.* 2.
- 1850 COHEN, DANIEL WHITAKER, M.D., South Bank, North Down lane, Bideford, Devon.
- 1854 COLLINS, FREDERICK, M.D., Wanstead Lodge, Essex.
- 1865 COOPER, ALFRED, Surgeon to the Lock Hospital; Assistant Surgeon to St. Mark's Hospital; Surgeon to the West London Hospital; 9, Henrietta street, Cavendish square.
- 1843 †COOPER, WILLIAM WHITE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9. V.P. 1873-4. *Lib. Com.*
- 1868 CORNISH, WILLIAM ROBERT, Surgeon-Major, Madras Army; Sanitary Commissioner for Madras; Secretary to the Inspector-General, Indian Medical Department.
- 1860 *CORRY, THOMAS CHARLES STEUART, M.D., Surgeon to the Belfast General Dispensary; 146, Donegall Pass, Belfast.
- 1853 CORY, WILLIAM GILLETT, M.D.
- 1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 17, Harley street, Cavendish square.
- 1860 †COUPER, JOHN, Surgeon to, and Lecturer on Surgery at, the London Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street. C. 1876.
- 1877 COUPLAND, SIDNEY, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; 7, Nottingham place.

Elected

- 1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 19, George street, Hanover square.
- 1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D. C. 1853-4.
- 1868 CRAWFORD, THOMAS, M.D., Deputy Inspector-General of Hospitals (India); Umbalah, Punjaub.
- 1873 CREIGHTON, CHARLES, M.B., Demonstrator of Anatomy, University of Cambridge, Anatomical Museum, Cambridge.
- 1869 *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.
- 1874 CRIPPS, WILLIAM HARRISON, Surgical Registrar to St. Bartholomew's Hospital; Surgeon to the Great Northern Hospital; Assistant Surgeon to the Royal Free Hospital; 6, Stratford place, Oxford street. *Trans.* 1.
- 1847 †CRITCHETT, GEORGE, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the Middlesex Hospital; 21, Harley street, Cavendish square. C. 1865. V.P. 1872. *Trans.* 1.
- 1868 CROFT, JOHN, Surgeon to, and Lecturer on Operative Surgery at, St. Thomas's Hospital; 61, Brook street, Grosvenor square. *Lib. Com.*
- 1862 CROMPTON, SAMUEL, M.D., Physician to the Salford Royal Hospital and Dispensary; 24, St. Ann's square, Manchester.
- 1837 CROOKES, JOHN FARRAR, Grimethorpe, Tunbridge Wells.
- 1860 CROSS, RICHARD, M.D., Carlton House, Belmont road, Scarborough.
- 1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.
- 1849 *CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.
- 1879 CUMBERBATCH, A. ELKIN, Demonstrator of Anatomy at St. Bartholomew's Hospital; 17, Queen Anne street.

Elected

- 1846 CURLING, HENRY, Consulting Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.
- 1837 †CURLING, THOMAS BLIZARD, F.R.S., Consulting-Surgeon to the London Hospital; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Trans.* 13. *Pro.* 1. *Sci. Com.* 1.
- 1873 CURNOW, JOHN, M.D., Professor of Anatomy at King's College, London, and Assistant Physician to King's College Hospital; Examiner in Anatomy at the University of London; 3, Warwick street, Cockspur street.
- 1847 CURREY, JOHN EDMUND, M.D., Lisimore, County Waterford.
- 1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.
- 1852 CUTLER, THOMAS, M.D., Spa, Belgium.
- 1872 DALBY, WILLIAM BARTLETT, M.B., Lecturer on Aural Surgery at St. George's Hospital; 18, Savile row. *Trans.* 2.
- 1836 *DANIEL, JAMES STOCK, Ramsgate, Kent.
- 1877 DARBISHIRE, SAMUEL DUKINFIELD, M.B., Physician to the Radcliffe Infirmary, Oxford.
- 1848 DAUBENY, HENRY, M.D., San Remo, Italy.
- 1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 49, Rodney street, Liverpool.
- 1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.
- 1852 DAVIES, WILLIAM, M.D., 18, Gay street, Bath.
- 1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Assistant Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 1.
- 1852 DAVIS, JOHN HALL, M.D., Physician Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Physician to the Royal Maternity Charity, and Consulting Physician-Accoucheur to the St. Pancras Infirmary; Examiner in Obstetric Medicine at the University of London; 24, Harley street, Cavendish square. C. 1869-70.

Elected

- 1878 DAVY, RICHARD, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 33, Welbeck street, Cavendish square.
- 1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.
- 1878 DENT, CLINTON THOMAS, 29, Chesham Street, Belgrave square.
- 1846 *DENTON, SAMUEL BEST, M.D., Ivy Lodge, Hornsea, Hull.
- 1859 †DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Medicine at St. George's Hospital, and Senior Physician to the Hospital for Sick Children; Examiner in Medicine at the University of London; 9, Chesterfield street, Mayfair. C. 1874-5. *Trans.* 13. *Sci. Com.* 3.
- 1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Consulting Ophthalmic Surgeon to the Asylum for Idiots; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Lib. Com.* *Trans.* 4.
- 1862 DOBELL, HORACE B., M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest, City road; 84, Harley street. *Trans.* 2.
- 1845 DODD, JOHN.
- 1879 DONKIN, HORATIO, M.B., 60, Upper Berkeley street, Portman square.
- 1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to Out-Patients, Samaritan Hospital; Pathological Assistant to the Museum of the Royal College of Surgeons of England; 51, Seymour street, Portman square.
- 1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to the London Hospital; 39, Welbeck street, Cavendish square. *Trans.* 2.
- 1867 DRAGE, CHARLES, M.D., Hatfield, Herts.
- 1853 DRUITT, ROBERT, F.R.C.P. [8, Strathmore gardens, Kensington Mall.] *Trans.* 2.

Elected

- 1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 17, Woburn place, Russell square.
- 1865 DUCKWORTH, DYCE, M.D., Assistant-Physician to, and Lecturer on Skin Diseases at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. *Trans.* 1.
- 1876 DUDLEY, WILLIAM LEWIS, M.D., Physician to the City Dispensary; 125, Cromwell road, South Kensington.
- 1845 DUFF, GEORGE, M.D., High street, Elgin.
- 1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.
- 1874 DUKA, THEODORE, M.D., [Surgeon-Major, H.M.'s Bengal Army]; 38, Montagu square.
- 1871 DUKE, BENJAMIN, 1, Cavendish terrace, Clapham Common.
- 1871 *DUKES, CLEMENT, M.D. and B.S., Horton crescent, Rugby, Warwickshire.
- 1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.
- 1877 DUNCAN, JAMES MATTHEWS, M.D., LL.D. Ed., F.R.S. Ed., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Bartholomew's Hospital; 71, Brook street, Grosvenor square.
- 1861 DU PASQUIER, CLAUDIUS FRANCIS, Surgeon-Apothecary to H.M. the Queen, and to the Household of H.R.H. the Prince of Wales.
- 1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. *Sci. Com.* 1. *Lib. Com.* *Trans.* 5.
- 1874 DURHAM, FREDERIC, M.B., Surgical Registrar to Guy's Hospital; 38, Brook street, Grosvenor square.
- 1843 DURRANT, CHRISTOPHER MERCER, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.
- 1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

Elected

- 1836 EARLE, JAMES WILLIAM, late of Norwich.
- 1868 EASTES, GEORGE, M.B. Lond., Surgeon-Accoucheur to the Western General Dispensary; 69, Connaught street, Hyde park square.
- 1824 EDWARDS, GEORGE.
- 1823 EGBERTON, CHARLES CHANDLER, Kendall Lodge, Epping.
- 1869 ELAM, CHARLES, M.D., Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 75, Harley street, Cavendish square.
- 1861 *ELLIOT, ROBERT, M.D., Physician to the Fever Hospital and to the Dispensary, Carlisle; Coroner for Carlisle; 35, Lowther street, Carlisle.
- 1848 ELLIS, GEORGE VINEY, late Professor of Anatomy in University College, London; Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.
- 1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.
- 1854 *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.
- 1842 †ERICHSEN, JOHN ERIC, F.R.S., *President*, Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; 6, Cavendish place, Cavendish square. C. 1855-6. V.P. 1868. P. 1879. *Lib. Com. Trans.* 2.
- 1874 EVANS, GEORGE HENRY, M.D.
- 1877 EWART, WILLIAM, M.B., 33, Curzon street, May Fair.
- 1875 *FAGAN, JOHN, Surgeon to the Belfast Hospital for Sick Children; 11, College square north, Belfast.
- 1864 FAGGE, CHARLES HILTON, M.D., Senior Assistant Physician to, and Lecturer on Pathology at, Guy's Hospital; Examiner in Medicine at the University of London; 11, St. Thomas's street, Southwark. *Sci. Com.* 1. *Lib. Com. Trans.* 6.
- 1869 FAIRBANK, FREDERICK ROYSTON, M.D., 46, Hallgate, Doncaster.

Elected

- 1858 FALCONER, RANDLE WILBRAHAM, M.D., Consulting Physician to the Royal United Hospital, Bath; 22, Bennett street, Bath.
- 1862 FARQUHARSON, ROBERT, M.D., Assistant Physician to, and Lecturer on Materia Medica at, St. Mary's Hospital; Physician to the Belgrave Hospital for Children; 23, Brook street, Grosvenor square. *Lib. Com.*
- 1844 †FARRE, ARTHUR, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Physician-Accoucheur to H.R.H. the Princess of Wales; 18, Albert Mansions, Victoria street, Westminster. C. 1857. V.P. 1864. *Sci. Com. 1. Lib. Com.*
- 1872 FAYREER, SIR JOSEPH, K.S.I., M.D., F.R.S. Edin., Honorary Physician to H.M. the Queen, and to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Surgeon-General, late Bengal Medical Service; Examining Medical Officer to the Secretary of State for India in Council; President of the Indian Medical Board; 16, Granville place, Portman square.
- 1872 *FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; 16, Old Elvet, Durham.
- 1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. *Trans. 3.*
- 1852 *FIELD, ALFRED GEORGE.
- 1849 FINCHAM, GEORGE TUPMAN, M.D., Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.
- 1879 FINLAY, DAVID WHITE, M.D., Assistant Physician to the Middlesex Hospital; 21, Montagu street, Portman square.
- 1866 FISH, JOHN CROCKETT, B.A., M.B. Camb., Assistant Physician to the West London Hospital; 92, Wimpole street, Cavendish square.
- 1860 FITZGERALD, THOMAS GEORGE, Surgeon-Major. [6, Whitehall yard.]

Elected

- 1866 FITZPATRICK, THOMAS, M.D., M.A., Dublin; Physician to the Western General Dispensary; 30, Sussex gardens, Hyde park.
- 1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 43, Clarendon square, Leamington. *Trans.* 1.
- 1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.
- 1877 FONMARTIN, HENRY DE, M.D., House Physician, Seamen's Hospital, Greenwich.
- 1848 †FORBES, JOHN GREGORY, 82, Oxford terrace, Hyde park. C. 1868-9. *Lib. Com. Trans.* 3.
- 1852 †FORSTER, JOHN COOPER, *Treasurer*, Surgeon to Guy's Hospital; Examiner in Surgery at the University of London; 29, Upper Grosvenor street. C. 1868-9. S. 1873-5. V.P. 1877-78. T. 1879. *Pro.* 1.
- 1877 *FORTESCUE, GEORGE, M.B., late Surgeon to the Sydney Infirmary; 6, Lyons terrace, Sydney, New South Wales.
- 1865 FOSTER, BALTHAZAR WALTER, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 16, Temple row, Birmingham.
- 1859 FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary, and Lecturer on Medicine at the Bristol School of Medicine; Church House, Clifton, Gloucestershire.
- 1858 FOX, WILSON, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Physician in Ordinary to H.R.H. the Duke of Edinburgh; Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 67, Grosvenor street. C. 1875-6. *Lib. Com. Trans.* 3.
- 1871 FRANK, PHILIP, M.D., Cannes, France.
- 1843 FRASER, PATRICK, M.D. C. 1866.
- 1868 FREEMAN, WILLIAM HENRY, 21, St. George's square, South Belgravia.

Elected

- 1836 †FRENCH, JOHN GEORGE, 10, Cunningham place, Maida hill. C. 1852-3.
- 1876 FURNER, WILLOUGHBY, 111, King's road, Brighton.
- 1864 *GAIRDNER, WILLIAM TENNANT, M.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary; 225, St. Vincent street, Glasgow.
- 1874 GALABIN, ALFRED LEWIS, M.A., M.D., Assistant Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; Assistant-Physician to the Hospital for Sick Children; 14, St. Thomas's street, Southwark. *Trans.* 2.
- 1865 GANT, FREDERICK JAMES, Senior Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde park. *Trans.* 2.
- 1867 GARLAND, EDWARD CHARLES, L.R.C.P. Edin., Yeovil, Somerset.
- 1867 GARLIKE, THOMAS W., Malvern Villa, Churchfield road, Ealing.
- 1854 GARROD, ALFRED BARING, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. *Trans.* 8.
- 1851 †GASKOIN, GEORGE, Surgeon to the British Hospital for Diseases of the Skin; 7, Westbourne park. C. 1875-6. *Trans.* 2.
- 1819 GAULTER, HENRY.
- 1848 †GAY, JOHN, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 10, Finsbury place south. C. 1874-5.
- 1866 GEE, SAMUEL JONES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Physician to the Hospital for Sick Children; 54, Harley street, Cavendish square. *Sci. Com.* 1. *Lib. Com.* *Trans.* 1.
- 1821 *GEORGE, RICHARD FRANCIS.

Elected

- 1878 GERVIS, HENRY, M.D., Obstetric Physician to, and Lecturer on Obstetric Medicine at, St. Thomas's Hospital; Physician to the Royal Maternity Charity; 40, Harley street, Cavendish square.
- 1877 GODLEE, RICKMAN JOHN, Assistant-Surgeon to University College Hospital; and Demonstrator of Anatomy at University College; 22, Henrietta street, Cavendish square.
- 1870 GODSON, CLEMENT, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; 8, Upper Brook street, Grosvenor square.
- 1867 GOODEVE, EDWARD, M.B., Hon. Physician to H.M. the Queen; late Surgeon-Major, H.M.'s Bengal Army; Drimagh, Stoke Bishop, near Bristol.
- 1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Trans.* 2.
- 1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to, and Lecturer on Anatomy at, the Westminster Hospital; 16, Queen Anne street, Cavendish square.
- 1873 GOWERS, WILLIAM RICHARD, M.D., Assistant Professor of Clinical Medicine at University College, and Assistant-Physician to University College Hospital; 50, Queen Anne street. *Trans.* 6.
- 1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury square.
- 1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; Crawleydown park, Worth, Sussex. C. 1863.
- 1868 GREEN, T. HENRY, M.D., Physician to, and Lecturer on Pathology at, Charing Cross Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square.
- 1875 GREENE, WILLIAM T., M.A., M.D., Moira House, Peckham rye.

Elected

- 1875 GREENFIELD, W. S., M.D., Professor Superintendent at the Brown Institution; Assistant Physician to, and Lecturer on Morbid Anatomy at, St. Thomas's Hospital; 15, Palace road, Albert Embankment. *Sci. Com.* 1.
- 1843 †GREENHALGH, ROBERT, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 72, Grosvenor street. C. 1871-2. *Trans.* 1.
- 1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Physician to the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; 14A, Manchester square. C. 1876-7. *Trans.* 3.
- 1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; Assistant-Physician to the Victoria Hospital for Children; 6, Curzon street, Mayfair.
- 1852 GROVE, JOHN, Spring Grove, Hampton, Middlesex.
- 1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris. *Lib. Com.*
- 1849 GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., F.R.S., Physician-Extraordinary to the Queen; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. V.P. 1874. *Trans.* 4.
- 1837 GULLY, JAMES MANBY, M.D.
- 1854 HABERSHON, SAMUEL OSBORNE, M.D., Physician to Guy's Hospital; 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. *Trans.* 3.
- 1849 HAILEY, HAMMETT, F.L.S., Tickford Lodge, Newport Pagnell, Bucks.
- 1870 HAMILTON, ROBERT, Surgeon to the South Hospital, Liverpool; 1 Prince's road, Liverpool.
- 1838 †HANCOCK, HENRY, Consulting Surgeon to the Charing Cross Hospital, and to the Royal Westminster Ophthalmic Hospital; Standen House, Chute, Wilts. C. 1851. V.P. 1869.

Elected

- 1874 **HARDIE, GORDON KENMURE, M.D.**, Deputy Inspector General of Hospitals; Florence road, Ealing, and Duff House, Banff, N.B.
- 1836 **HARDING, JOHN FOSSE**, Ulverstone House, Uckfield, Sussex. C. 1858-9.
- 1856 **HARE, CHARLES JOHN, M.D.**, late Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 57, Brook street, Grosvenor square. C. 1873-4.
- 1857 **HARLEY, GEORGE, M.D., F.R.S.** 25, Harley street, Cavendish square. C. 1871-2. *Trans.* 1. *Sci. Com.* 2.
- 1864 **HARLEY, JOHN, M.D., F.L.S.**, Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 39, Brook street, Grosvenor square. S. 1875-7. C. 1879. *Trans.* 8.
- 1866 **HARPER, PHILIP H.**, 30, Cambridge street, Hyde park.
- 1859 **HARRIS, FRANCIS, M.D., F.L.S.**, 24, Cavendish square.
- 1872 **HARRIS, WILLIAM H., M.D.**, Professor of Midwifery and Diseases of Women and Children, Madras Medical College, Madras.
- 1870 **HARRISON, REGINALD**, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Surgery at the School of Medicine; 38, Rodney street, Liverpool.
- 1854 **HAVILAND, ALFRED**, Medical Officer of Health for the combined Districts of Northamptonshire; Northampton.
- 1870 **HAWARD, J. WARRINGTON**, Assistant-Surgeon to St. George's Hospital; Surgeon to the Hospital for Sick Children; 5, Montagu street, Portman square. *Trans.* 1.
- 1828 †**HAWKINS, CÆSAR HENRY, F.R.S.**, Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.
- 1838 †**HAWKINS, CHARLES**, Inspector of Anatomical Schools in London; 27, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Lib. Com. Trans.* 2.

Elected

- 1848 HAWKESLEY, THOMAS, M.D., Consulting Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 31, Grosvenor street.
- 1875 HAYES, THOMAS CRAWFORD, M.D., Assistant-Physician-Accoucheur and Assistant-Physician for Diseases of Women and Children to King's College Hospital; 17, Clarges street, Piccadilly.
- 1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.
- 1861 HAYWARD, WILLIAM HENRY, Church House, Oldbury, Worcestershire.
- 1848 *HEALE, JAMES NEWTON, M.D., Medecroft, Winchester, Hants.
- 1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. *Lib. Com. Trans.* 1.
- 1850 HEATON, GEORGE, M.D., Boston, U.S.
- 1874 *HEATON, JOHN DEAKIN, M.D., Senior Physician to the Leeds General Infirmary, and Lecturer on Medicine at the Leeds School of Medicine; Claremont, Leeds.
- 1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.
- 1877 HERMAN, GEORGE ERNEST, M.R.C.P., Assistant Obstetric Physician to the London Hospital; 20, Finsbury square.
- 1877 HERON, GEORGE ALLAN, M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Assistant Physician to the West London Hospital for the Paralysed and Epileptic; 40, Margaret street, Cavendish square.
- 1843 HEWETT, PRESCOTT GARDNER, F.R.S., Serjeant-Surgeon-Extraordinary to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; Corresponding Member of the Academy of Medicine, and of the "Société de Chirurgie," Paris; 1, Chesterfield street, Mayfair. C. 1859. V.P. 1866-7. *Sci. Com.* 1. *Lib. Com. Trans.* 7.

Elected

- 1855 HEWITT, GRAILY, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876. *Lib. Com.*
- 1872 HEYN, JULIUS CHARLES WILLIAM, M.D., 42, Westbourne terrace, Hyde park.
- 1873 HIGGENS, CHARLES, Assistant Ophthalmic Surgeon to Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 1.
- 1862 HILL, M. BERKELEY, M.B. Lond., Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Surgeon to the Lock Hospital; 55, Wimpole street, Cavendish square. C. 1878-9.
- 1867 HILL, SAMUEL, M.D., 22, Mecklenburgh square.
- 1859 HIRD, FRANCIS, Senior Surgeon to the Charing Cross Hospital; 13, Old Burlington street.
- 1861 *HOFFMEISTER, WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Clifton House, Cowes, Isle of Wight.
- 1872 HOGG, FRANCIS ROBERTS, M.D., India.
- 1843 †HOLDEN, LUTHER, Surgeon to St. Bartholomew's Hospital; Consulting Surgeon to the Metropolitan Dispensary; Surgeon to the Foundling Hospital; 65, Gower street, Bedford square. C. 1859. L. 1865. V.P. 1874. *Lib. Com.*
- 1879 HOLLAND, PHILIP ALEXANDER, M.A., St. Bartholomew's Hospital.
- 1868 HOLLIS, WILLIAM AINSLIE, M.A., M.B., Camb., Assistant-Physician to the Sussex County Hospital; 10, Old Steyne, Brighton.
- 1861 HOLMAN, WILLIAM HENRY, M.B. Lond., 68, Adelaide road, South Hampstead.
- 1856 HOLMES, TIMOTHY, M.A. Camb., *Secretary*, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; Surgeon in Chief to the Metropolitan Police Force; 18, Great Cumberland place, Hyde park. C. 1869-70. L. 1873-7. S. 1878. *Sci. Com.* 1. *Lib. Com. Trans.* 6.

Elected

- 1846 †HOLT, BARNARD WIGHT, *Vice-President*, Consulting Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3. V.P. 1879.
- 1846 †HOLTHOUSE, CARSTEN, 26, Guilford street, Russell square. C. 1863. *Lib. Com.*
- 1878 HOOD, DONALD WILLIAM CHARLES, M.B., 43, Green street, Park lane.
- 1879 HOOD, FRANCIS E. C., Banstead Asylum, near Sutton, Surrey.
- 1878 HOUGHTON, WALTER B., M.D., Assistant Physician to Charing Cross Hospital; 26, Cavendish square.
- 1865 HOWARD, BENJAMIN, M.D., late Lecturer on Operative Surgery at the University of New York, and Surgeon to the Long Island College Hospital, New York; 116, East Nineteenth street, New York, U.S.
- 1865 HOWARD, EDWARD, M.D.
- 1874 HOWSE, HENRY GREENWAY, M.S. Lond., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 10, St. Thomas's street, Southwark. *Sci. Com.* 1. *Trans.* 2.
- 1877 *HUDSON, ROBERT SAMUEL, M.D., 58, West-end, Redruth, Cornwall.
- 1857 HULKE, JOHN WHITAKER, F.R.S., *Librarian*, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879. *Sci. Com.* 1. *Lib. Com.* *Trans.* 8.
- 1857 HULME, EDWARD CHARLES, Woodbridge road, Guildford. *Trans.* 1.
- 1844 †HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.
- 1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Human Anatomy and Physiology in the Cambridge University Medical School; Cambridge. *Trans.* 6.

Elected

- 1873 HUNTER, WILLIAM GUYER, M.D., Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-Major, Bombay Army, Bombay.
- 1849 HUSSEY, EDWARD LAW, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 8, St. Aldate's, Oxford. *Trans.* 1.
- 1856 HUTCHINSON, JONATHAN, Senior Surgeon to the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. *Lib. Com. Trans.* 9. *Pro.* 2.
- 1820 HUTCHINSON, WILLIAM, M.D.
- 1840 †HUTTON, CHARLES, M.D., Consulting Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.
- 1866 ILES, FRANCIS HENRY WILSON, M.D., Watford, Herts.
- 1847 IMAGE, WILLIAM EDMUND, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.
- 1856 INGLIS, CORNELIUS, M.D., Cairo. [Athenæum Club, Pall Mall.]
- 1876 IRVINE, JAMES PEARSON, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, the Charing Cross Hospital; 3, Mansfield street, Portland place.
- 1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.
- 1841 †JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.
- 1863 JACKSON, THOMAS VINCENT, Surgeon to the South Staffordshire General Hospital; Darlington st., Wolverhampton.
- 1841 JACOBOVICS, MAXIMILIAN MORITZ, M.D., Vienna.
- 1825 JAMES, JOHN B., M.D.
- 1840 *JENKS, GEORGE SAMUEL, M.D., 18, Circus, Bath.

Elected

- 1851 JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Member of the Senate of the University of London; 63, Brook street, Grosvenor square. C. 1864. V.P. 1875. *Trans.* 3.
- 1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Gènevois."
- 1847 †JOHNSON, GEORGE, M.D., F.R.S., *Librarian*, Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. L. 1878-9. *Lib. Com. Trans.* 10.
- 1868 JOHNSTON, WILLIAM, M.D., 44, Princes square, Hyde park.
- 1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Brighton Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Trans.* 1.
- 1876 JONES, LESLIE, M.D., 3, Brighton Parade, Blackpool, Lancashire.
- 1875 *JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., 1, Gresham buildings, Basinghall street.]
- 1837 †JONES, THOMAS WILLIAM, M.D., Bylocks, Enfield Highway. C. 1858.
- 1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.
- 1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; 22, Colmore row, Birmingham.
- 1816 *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.

Elected

- 1872 KELLY, CHARLES, M.D., Professor of Hygiene at King's College, London.
- 1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.
- 1877 *KHORY, RUSTOMJEE NASERWANJEE, M.D. Brussels; Physician to the Parell Dispensary, Bombay; Lecturer to Native Midwives, Grant Medical College, Bombay.
- 1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens, Bayswater, W.
- 1851 KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Trans.* 1. *Sci. Com.* 1.
- 1855 LANE, JAMES ROBERT, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; Surgeon to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1870. *Lib. Com.* *Trans.* 1.
- 1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1849-50. V.P. 1865.
- 1865 LANGTON, JOHN, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 2, Harley street, Cavendish square. *Lib. Com.*
- 1873 *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.
- 1841 *LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.
- 1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.
- 1816 LAWRENCE, G. E.

Elected

- 1843 †LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Trans.* 13. *Pro.* 2. *Sci.Com.* 1.
- 1877 LEESON, ARTHUR EDMUND, M.A., M.D., 45, Devonshire street, Portland Place.
- 1869 LEGG, JOHN WICKHAM, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, St. Bartholomew's Hospital; 47, Green street, Park lane. *Trans.* 2.
- 1836 LEIGHTON, FREDERICK, M.D.
- 1872 LIEBREICH, RICHARD, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; 16, Albemarle street, Piccadilly.
- 1806 LIND, JOHN, M.D.
- 1878 LISTER, JOSEPH, F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Clinical Surgery at King's College, London; and Surgeon to King's College Hospital; 12, Park crescent, Regent's park.
- 1872 *LITTLE, DAVID, M.D., Surgeon to the Royal Eye Hospital, Manchester; 21, St. John's street, Manchester.
- 1871 LITTLE, LOUIS STROMEYER, Shanghai, China.
- 1870 LIVINGSTON, JOHN, M.D., New Barnet, Hertfordshire.
- 1819 LLOYD, ROBERT, M.D.
- 1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.
- 1860 LONGMORE, THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.
- 1836 LÖWENFELD, JOSEPH S., M.D., Berbice.
- 1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.
- 1877 LOWNE, BENJAMIN THOMPSON, Lecturer on Physiology, Middlesex Hospital Medical School; 65, Cambridge gardens, Notting hill, W.

Elected

- 1852 LUKE, JAMES, F.R.S., Consulting Surgeon to the London Hospital; Fingest Grove, High Wycombe, Bucks. C. 1858. *Trans.* 4.
- 1879 LYELL, ROBERT, Assistant Surgeon to the Middlesex Hospital; 26, Harley street, Cavendish square.
- 1857 LYON, FELIX WILLIAM, M.D., 49, Albany street, Leith, near Edinburgh.
- 1867 MABERLY, GEORGE FREDERICK
- 1873 MACCARTHY, JEREMIAH, M.A., Surgeon to, and Lecturer on Physiology at, the London Hospital; 26, Finsbury square.
- 1867 MAC CORMAC, WILLIAM, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. *Trans.* 1.
- 1862 *M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Hospital; 14, Lower Pembroke street, Dublin. *Trans.* 2.
- 1846 M'EWEN, WILLIAM, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.
- 1866 MACGOWAN, ALEXANDER THORBURN, Vyvyan House, Clifton, near Bristol.
- 1823 †MACILWAIN, GEORGE, Consulting Surgeon to the Finsbury Dispensary, Matching, Harlow, Essex. C. 1829-30. V.P. 1848. *Lib. Com. Trans.* 1.
- 1822 MACINTOSH, RICHARD, M.D.
- 1859 *M'INTYRE, JOHN, M.D., Odiham, Hants.
- 1873 MACKELLAR, ALEXANDER OBERLIN, M.S.I., Assistant Surgeon, St. Thomas's Hospital; Albert Embankment, Westminster Bridge.
- 1876 MACKEY, EDWARD, M.D., 22, Norfolk road, Brighton.
- 1854 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.
- 1879 MACLAGAN, THOMAS JOHN, M.D., 9, Cadogan place, Belgrave square.
- 1860 MACLEAN, JOHN, M.D., 24, Portman street, Portman square.

Elected

- 1849 MACLURE, DUNCAN MACLACHLAN, M.B., Assistant-Physician to the National Hospital for the Paralyse and Epileptic ; 34, Harley Street, Cavendish square.
- 1876 MACNAMARA, CHARLES, Surgeon to the Westminster Hospital ; Surgeon Major Bengal Medical Service ; late Examiner in Surgery at the Calcutta University ; 13, Grosvenor street.
- 1842 MACNAUGHT, JOHN, M.D., 74, Huskisson street, Liverpool.
- 1876 MALLAM, BENJAMIN, Meadow Side, Leacroft road, Staines.
- 1855 MARCET, WILLIAM, M.D., F.R.S. ; Villa Bianca, Cannes. [2, Bolton street, Piccadilly.] C. 1871. *Sci. Com.* 1. *Lib. Com. Trans.* 3.
- 1867 MARSH, F. HOWARD, Assistant-Surgeon to St. Bartholomew's Hospital ; Assistant Surgeon to the Hospital for Sick Children, Great Ormond street ; 36, Bruton street, Berkeley square. *Trans.* 2.
- 1838 MARSH, THOMAS PARR, M.D.
- 1851 MARSHALL, JOHN, F.R.S., Professor of Anatomy to the Royal Academy of Arts ; Professor of Surgery in University College, London, and Surgeon to University College Hospital ; 10, Savile row, Burlington gardens. C. 1866. V.P. 1875-76. *Trans.* 2.
- 1864 MASON, FRANCIS, Surgeon to, and Lecturer on Anatomy at, St. Thomas's Hospital ; 5, Brook street, Grosvenor square. *Trans.* 1.
- 1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary ; Bradford, Yorkshire. *Trans.* 1.
- 1870 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital ; 27, George street, Hanover square. *Lib. Com.*
- 1865 MEDWIN, AARON GEORGE, M.D., Dental Surgeon to the Royal Kent Dispensary, 11, Montpelier row, Blackheath, Kent.
- 1867 MEREDYTH, COLOMIATI, M.D., 10, George street, Hanover square.
- 1874 MERRIMAN, JOHN J., 45, Kensington square.
- 1815 MEYER, AUGUSTUS, M.D., St. Petersburg.

Elected

- 1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital; 19, Temple row, Birmingham.
- 1854 MIDDLESHIP, EDWARD ARCHIBALD.
- 1873 MILNER, EDWARD, Surgeon to the Lock Hospital; 32, New Cavendish street, Portland place.
- 1844 †MONTEFIORE, NATHANIEL, 18, Portman square.
- 1836 MOORE, GEORGE, M.D., Hastings.
- 1873 MOORE, NORMAN, M.D., Warden of the College and Lecturer on Comparative Anatomy, Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; the College, St. Bartholomew's Hospital.
- 1861 MOREHEAD, CHARLES, M.D., Hon. Surgeon to H.M. the Queen; Deputy-Inspector General of Hospitals; 11, North manor place, Edinburgh.
- 1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. *Lib. Com. Trans. 1.*
- 1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Owens College, Manchester; 1, St. Peter's square, Manchester.
- 1878 MORGAN, JOHN HAMMOND, M.A., 12, Chapel street, Park lane.
- 1874 MORRIS, HENRY, M.A. Lond., Surgeon to, and Lecturer on Clinical Surgery at, the Middlesex Hospital; 2, Mansfield street, Portland place. *Trans. 2.*
- 1879 MORRIS, MALCOLM ALEXANDER, Joint Lecturer on Skin Diseases at St. Mary's Hospital; 63, Montagu square.
- 1851 MOUAT, FREDERIC JOHN, M.D., Deputy Inspector-General of Hospitals; Medical Inspector to the Local Government Board; and Member of the Senate of the University of Calcutta; 12, Durham villas, Kensington.
- 1868 MOXON, WALTER, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; 6, Finsbury Circus. *Trans. 1.*
- 1875 MURPHY, WILLIAM KIRKPATRICK, M.A., M.D., 29, Queen Anne street, Cavendish square.

Elected

- 1873 MURRAY, IVOR, M.D., F.R.S. Ed., 8, Huntress Row, Scarborough.
- 1863 MYERS, ARTHUR BOWEN RICHARDS, Surgeon to the 1st Battalion, Coldstream Guards; Hospital, Vincent square, Westminster. C. 1878-9. *Lib. Com.*
- 1876 NAPIER, WILLIAM DONALD, 22, George street, Hanover square.
- 1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.
- 1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park. *Lib. Com.*
- 1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; 4, Wimpole street, Cavendish square.
- 1843 †NEWTON, EDWARD, 85, Gloucester terrace, Hyde Park. C. 1863-4.
- 1868 NICHOLLS, JAMES, M.D., Duke street, Chelmsford, Essex.
- 1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants.
- 1847 *NOURSE, WILLIAM EDWARD CHARLES, late Surgeon to the Brighton Children's Hospital; Bouverie House, Mount Radford, Exeter.
- 1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.
- 1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.
- 1847 O'CONNOR, THOMAS, March, Cambridgeshire.
- 1843 †O'CONNOR, WILLIAM, M.D., Senior Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.
- 1858 OGLE, JOHN WILLIAM, M.D., Consulting-Physician to St. George's Hospital; 30, Cavendish square. C. 1873. *Trans.* 4.
- 1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; The Elms, Duffield road, Derby.

Elected

- 1860 OGLE, WILLIAM, M.D., Medical Officer of Health for East Hertfordshire; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. *Lib. Com. Trans.* 4.
- 1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament Street].
- 1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Lincoln.
- 1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 7, Brook street, Hanover square. *Trans.* 4.
- 1877 ORMERON, JOSEPH ARDERNE, M.B., Casualty Physician to St. Bartholomew's Hospital; 25, Upper Wimpole street.
- 1875 OSBORN, SAMUEL, 17, Gresham park, Brixton, and 10, Maddox street, Regent street.
- 1879 OWEN, EDMUND, Assistant Surgeon to St. Mary's Hospital; 49, Seymour street, Portman square.
- 1874 PAGE, HERBERT WILLIAM, M.A., M.C. Cantab., Assistant Surgeon to, and Lecturer on Operative and Practical Surgery at, St. Mary's Hospital; 28, New Cavendish street. *Trans.* 1.
- 1847 *PAGE, WILLIAM BOUSFIELD, Consulting Surgeon to the Cumberland Infirmary, Carlisle. *Trans.* 2.
- 1840 †PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Member of the Senate of the University of London; 1, Harewood place, Hanover square. C. 1848-49. V.P. 1861. T. 1867. P. 1875-76. *Sci. Com.* 1. *Lib. Com. Trans.* 11.
- 1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.
- 1847 PARKER, NICHOLAS, M.D., Paris.
- 1873 PARKER, ROBERT WILLIAM, Assistant-Surgeon East London Children's Hospital; 8, Old Cavendish-street. *Trans.* 2.
- 1841 PARKIN, JOHN, M.D., 5, Codrington place, Brighton.
- 1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 35, Grosvenor street.

Elected

- 1869 PAYNE, JOSEPH FRANK, M.B., Assistant-Physician to, and Lecturer on Forensic Medicine at, St. Thomas's Hospital; 78, Wimpole street. *Sci. Com.* 1. *Lib. Com.*
- 1845 †PEACOCK, THOMAS BEVILL, M.D., Consulting Physician to St. Thomas's Hospital; Consulting Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 20, Finsbury circus. S. 1855-6. V.P. 1867. C. 1869. *Trans.* 2.
- 1856 PEIRCE, RICHARD KING, 94, Addison road, Kensington.
- 1830 PELECHIN, CHARLES P., M.D., St. Petersburg.
- 1855 *PEMBERTON, OLIVER, Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 12, Temple row, Birmingham. *Trans.* 1.
- 1874 PENHALL, JOHN THOMAS, 5, Eversfield place, St. Leonard's, Sussex.
- 1870 PERRIN, JOHN BESWICK, Medical Tutor and late Demonstrator of Practical and Surgical Anatomy, Owen's College; The Homestead, Leigh, Lancashire.
- 1879 *PESIKAKA, HORMASJI DOSABHAI, L.R.C.P. Glasg., Marine Lines, Bombay.
- 1878 *PHILIPSON, GEORGE HARE, M.D., M.A. Cantab., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Infirmary; 7, Eldon square, Newcastle-upon-Tyne.
- 1852 PHILLIPS, RICHARD, 27, Leinster square, Bayswater. C. 1877.
- 1846 PHILP, FRANCIS RICHARD, M.D. [Colby House, Kensington.]
- 1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 13, South Eaton place, Eaton square. *Sci. Com.* 1. *Lib. Com.*
- 1841 †PITMAN, HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital, and to the Royal General Dispensary, St. Pancras; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Lib. Com.*

Elected

- 1871 POLLOCK, ARTHUR JULIUS, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.
- 1845 †POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1? *Trans.* 4.
- 1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square.
- 1871 POORE, GEORGE VIVIAN, M.D., Assistant-Physician to University College Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street. *Trans.* 1.
- 1846 POTTER, JEPHSON, M.D., F.L.S.
- 1842 POWELL, JAMES, M.D.
- 1867 POWELL, RICHARD DOUGLAS, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; Assistant-Physician to the Middlesex Hospital; 15, Henrietta street, Cavendish square. *Trans.* 1.
- 1867 POWER, HENRY, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Gt. Cumberland place, Hyde park. *Sci. Com.* 1. *Lib. Com.*
- 1857 PRIESTLEY, WILLIAM OVEREND, M.D., Consulting Physician-Accoucheur to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. *Sci. Com.* 1.
- 1869 PULLAR, ALFRED, M.D., Surgeon to the Kensington Dispensary; 1, Pembridge place, Bayswater.
- 1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 6, Stratford place, Oxford street. *Trans.* 2.
- 1878 PYE, WALTER, Assistant Surgeon to St. Mary's Hospital; 4, Sackville street, Piccadilly.
- 1877 PYE-SMITH, PHILIP HENRY, M.D., Assistant-Physician to, and Lecturer on Physiology at, Guy's Hospital; Examiner in Physiology at the University of London; 56, Harley street, Cavendish square.

Elected

- 1850 QUAIN, RICHARD, M.D., F.R.S., *Vice-President*, Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1. *Trans.* 1.
- 1835 †QUAIN, RICHARD, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to the Eye Infirmary attached to the Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Lib. Com.* *Trans.* 1. *Pro.* 2.
- 1852 RADCLIFFE, CHARLES BLAND, M.D., *Vice-President*, Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8. V.P. 1879.
- 1871 RALFE, CHARLES HENRY, M.D., M.A., Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square.
- 1857 RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.
- 1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital, Nottingham.
- 1869 READ, THOMAS LAURENCE, 57, Gloucester road [11, Peter-sham terrace], Queen's gate, South Kensington.
- 1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.
- 1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.
- 1857 REES, GEORGE OWEN, M.D., F.R.S., Consulting Physician to Guy's Hospital; 26, Albemarle street, Piccadilly. C. 1873. *Trans.* 1.
- 1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Physician-in-Ordinary to H.M.'s Household; Consulting-Physician to University College Hospital; 38, Grosvenor street. C. 1870.
- 1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

Elected

- 1852 RICHARDSON, CHRISTOPHER THOMAS, M.B.
- 1845 †RIDGE, BENJAMIN, M.D., 8, Mount street, Grosvenor square.
- 1863 RINGER, SYDNEY, M.D., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; Examiner in Materia Medica in the University of London; 15, Cavendish place, Cavendish square. *Trans.* 4.
- 1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital; 22, Finsbury square. *Trans.* 2.
- 1871 *ROBERTS, DAVID LLOYD, M.D., Physician to St. Mary's Hospital, Manchester; 23, St. John's street, Deansgate, Manchester.
- 1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics in University College, London; and Assistant Physician to University College Hospital; 53, Harley street, Cavendish square, W.
- 1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.
- 1873 ROBERTSON, WILLIAM H., M.D., Consulting Physician to the Devonshire Hospital and Buxton Bath Charity; Buxton, Derbyshire.
- 1843 RODEN, WILLIAM, M.D., Morningside, Kidderminster, Worcestershire.
- 1850 ROPER, GEORGE, M.D., Physician to the Eastern Division of the Royal Maternity Charity; Physician to the Royal Infirmary for Children and Women, Waterloo Bridge road; 6, West street, Finsbury circus. C. 1879.
- 1857 ROSE, HENRY COOPER, M.D., F.L.S., Surgeon to the Hampstead Dispensary; Penrose House, Hampstead. *Trans.* 1.
- 1849 ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com. Trans.* 1.

Elected

- 1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.
- 1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General Hospital; 91, New Hall street, Birmingham.
- 1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of Physiology in the University of Edinburgh.
- 1856 SALTER, S. JAMES A., F.R.S., F.L.S., Dental Surgeon to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City. C. 1871. *Trans.* 2.
- 1849 †SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3. *Lib. Com.*
- 1855 SANDERSON, JOHN BURDON, M.D., LL.D., F.R.S., Professor of Physiology at the University of London; Jodrell Professor of Human Physiology and Histology at University College, London; 26, Gordon square. C. 1869-70. *Sci. Com.* 2. *Lib. Com.* *Trans.* 2.
- 1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.
- 1879 SANGSTER, ALFRED, B.A., M.B., Lecturer on Skin Diseases at the Charing Cross Hospital; 7, Old Burlington street.
- 1847 †SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Sandywell park, Andoversford, Cheltenham.
- 1869 SANSOM, ARTHUR ERNEST, M.D., Assistant-Physician to the London Hospital; 30, Devonshire street, Portland place. *Trans.* 2.
- 1845 †SAUNDERS, EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square. C. 1872-3.
- 1834 SAUVAN, LUDWIG V., M.D., Warsaw.
- 1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; Examiner in Surgery at the University of London; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. *Sci. Com.* 4. *Lib. Com.* *Trans.* 5.
- 1873 SCOTT, JOHN MOORE JOHNSTON, M.D., Lurgan, County Armagh.

Elected

- 1861 *SCOTT, WILLIAM, M.D., Physician to the Huddersfield Infirmary ; Waverley House, Huddersfield.
- 1877 SEATON, EDWARD CATOR, M.D., Medical Officer of the Local Government Board ; 14, Gordon street, Gordon square.
- 1863 SEDGWICK, WILLIAM, 12, Park place, Upper Baker street.
Trans. 2.
- 1877 SEMON, FELIX, M.D., 59, Welbeck street, Cavendish square.
- 1875 SEMPLE, ROBERT HUNTER, M.D., Physician to the Bloomsbury Dispensary ; 8, Torrington square. *Sci. Com.* 1.
- 1873 *SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital ; the Barnfield, Exeter.
- 1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby.
Trans. 1.
- 1837 †SHARPEY, WILLIAM, M.D., F.R.S., LL.D., Member of the Senate of the University of London ; 50, Torrington square. C. 1848-9. V.P. 1862. *Lib. Com.*
- 1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital ; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Lib. Com. Trans.* 4.
- 1859 SIBLEY, SEPTIMUS WILLIAM, 7, Harley street, Cavendish square. *Sci. Com.* 1. *Trans.* 4.
- 1848 †SIEVEKING, EDWARD HENRY, M.D., Physician-Extraordinary to H.M. the Queen ; Physician-in-Ordinary to H.R.H. the Prince of Wales ; Physician to St. Mary's Hospital ; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. *Sci. Com.* 1. *Trans.* 2.
- 1871 SILVER, ALEXANDER, M.D., Physician to, and Lecturer on Clinical Medicine at, Charing Cross Hospital ; 2, Stafford street, Bond street.
- 1842 †SIMON, JOHN, C.B., D.C.L., F.R.S., Consulting Surgeon to St. Thomas's Hospital ; 40, Kensington square. C. 1854-5. V.P. 1865. *Trans.* 1.
- 1865 SIMS, J. MARION, M.D., Surgeon to the New York State Women's Hospital ; 267, Madison Avenue, New York.
- 1857 SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Nice.

Elected

- 1872 SMITH, GILBERT, M.A., M.B., Assistant-Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square.
- 1866 SMITH, HEYWOOD, M.A. M.D. Oxon., Physician to the Hospital for Women; Physician to the British Lying-in Hospital; 2, Portugal street, Grosvenor square.
- 1838 †SMITH, SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Lib. Com.*
- 1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-76. *Trans.* 3. *Sci. Com.* 2.
- 1864 *SMITH, THOMAS HECKSTALL, Rowlands, St. Mary Cray, Kent.
- 1847 SMITH, WILLIAM J., M.D., Consulting Physician to the Weymouth Infirmary; Greenhill, Weymouth, Dorsetshire.
- 1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.
- 1874 *SMITH, WILLIAM ROBERT, M.D., Physician to the Dispensary, Cheltenham; 15, Imperial square, Cheltenham.
- 1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.
- 1865 SOUTHEY, REGINALD, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 6, Harley street, Cavendish square. *Trans.* 1.
- 1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Alban's.
- 1874 SPARKS, EDWARD ISAAC, M.B., Mentone. *Trans.* 2.
- 1851 SPITTA, ROBERT JOHN, M.D. Lond., Clapham Common, Surrey. C. 1878-9. *Trans.* 1.
- 1875 SPITTA, EDMUND J., Ivy House, Clapham Common, Surrey.

Elected

- 1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board; Greenford House, Sutton, Surrey.
- 1842 †STEWART, ALEXANDER PATRICK, M.D., Consulting Physician to the Middlesex Hospital; 75, Grosvenor street. C. 1856-7. L. 1863-8. V.P. 1871-2.
- 1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.
- 1879 STIRLING, EDWARD CHARLES, Assistant Surgeon to St. George's Hospital; 60, Great Cumberland place, Hyde park.
- 1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.
- 1865 STOKES, WILLIAM, M.D., Examiner in Surgery, Queen's University, Ireland, and Surgeon to the Richmond Surgical Hospital; 5, Merrion square north, Dublin. *Trans.* 1.
- 1843 STOKES, ROBERT REEVE, Paris.
- 1858 †STREATFEILD, JOHN FREMLYN, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square. C. 1874-5. *Lib. Com.*
- 1876 STRETTON, WILLIAM HARRIS, M.D., Physician to the Farringdon Dispensary; 8, Suffolk place, Pall Mall East.
- 1871 STRONG, HENRY JOHN, M.D., 64, North End, Croydon.
- 1863 STURGES, OCTAVIUS, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Assistant-Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square. C. 1878-9.
- 1871 SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.
- 1860 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

Elected

- 1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Medicine at, the London Hospital, and Physician to the City of London Hospital for Diseases of the Chest; 9, Finsbury square. *Trans.* 1.
- 1855 SUTTON, JOHN MAULE, M.D., Medical Officer of Health, Oldham; 244, Great Clowes street, Broughton, Manchester.
- 1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.
- 1854 *SYMONDS, FREDERICK, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 35, Beaumont street, Oxford.
- 1878 *SYMPSON, THOMAS, Surgeon to the Lincoln County Hospital; 3, James street, Lincoln.
- 1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, Great Charles street, Birmingham. *Trans.* 1.
- 1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.
- 1875 TAY, WARREN, Surgeon to the London Hospital and Surgeon to the North Eastern Hospital for Children and the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.
- 1873 TAYLOR, FREDERICK, M.D., Assistant-Physician to Guy's Hospital; 15, St. Thomas's street, Southwark. *Trans.* 1.
- 1852 TAYLOR, ROBERT, 7, Lower Seymour street, Portman square.
- 1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's wood.
- 1859 TEGART, EDWARD, 49, Jermyn street, St. James's.
- 1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 6.
- 1862 THOMPSON, EDMUND SYMES, M.D., Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Trans.* 1. *Sci. Com.*

Elected

- 1857 THOMPSON, HENRY, M.D., Consulting Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.
- 1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 5.
- 1862 THOMPSON, REGINALD EDWARD, M.D., Assistant-Physician to the Hospital for Consumption, Brompton; 9, Cranley place, South Kensington. C. 1879. *Trans.* 2. *Sci. Com.* 1.
- 1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 83, Park street, Grosvenor square. *Trans.* 1.
- 1875 TIBBITS, HERBERT, F.R.C.P. Ed., Medical Superintendent of the National Hospital for the Paralysed and Epileptic; 30, New Cavendish street.
- 1848 †TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square.
- 1872 TOMES, CHARLES S., B.A., F.R.S., Lecturer on Anatomy and Physiology at the Dental Hospital; 37, Cavendish square. *Lib. Com.*
- 1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.
- 1871 *TREND, THEOPHILUS W., M.R.C.P. Edinb., Raeberry Lodge, Southampton.
- 1879 TREVES, FREDERICK, Assistant Surgeon to the London Hospital; 18, Gordon square.
- 1867 TROTTER, JOHN WILLIAM, Surgeon-Major, Coldstream Guards; Bossall Vicarage, York.
- 1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.
- 1864 TUFNELL, THOMAS JOLLIFFE, Consulting Surgeon to the City of Dublin Hospital; 58, Lower Mount street, Merrion square, Dublin. *Trans.* 1.

Elected

- 1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.
- 1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the London Hospital; 15, Finsbury square.
- 1873 TURNER, GEORGE BROWN, M.D., San Remo, Italy.
- 1876 VENN, ALBERT JOHN, M.D., Obstetric Physician to the Metropolitan Free Hospital; Assistant Physician to the Victoria Hospital for Children; 40, Brook street, Grosvenor square.
- 1870 VENNING, EDGCOMBE, late Surgeon, 1st Life Guards; 87, Sloane street.
- 1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 43, Weymouth street, Portland place.
- 1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy and to the French Hospital, Lisle street, Leicester square; 141, Regent street.
- 1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.
- 1854 WADDINGTON, EDWARD, Auckland, New Zealand.
- 1870 WADHAM, WILLIAM, M.D., Physician to, and Lecturer on Clinical Medicine at, St. George's Hospital; 14, Park lane.
- 1864 WAITE, CHARLES DERBY, M.B., Senior Physician to the Westminster General Dispensary; 3, Old Burlington street.
- 1868 *WALKER, ROBERT, L.R.C.P. Edinb., Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.
- 1867 *WALLIS, GEORGE, Corpus Buildings, Cambridge.
- 1873 WALSHAM, WILLIAM JOHNSON, C.M., Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital and to the Royal Hospital for Diseases of the Chest, City Road; 27, Weymouth street, Portland place.

Elected

- 1852 WALSH, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 37, Queen Anne street, Cavendish square. C. 1872. *Trans.* 1.
- 1851 WALTON, HAYNES, Senior Surgeon to St. Mary's Hospital, and to the Ophthalmic Department; 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.
- 1852 WANE, DANIEL, M.D., 20, Grafton street, Berkeley square.
- 1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.
- 1858 WARDELL, JOHN RICHARD, M.D., Calverley park, Tunbridge Wells.
- 1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.
- 1818 WARE, JOHN, Clifton Down, near Bristol.
- 1866 WARING, EDWARD JOHN, M.D., 49, Clifton gardens, Maida vale.
- 1877 WARNER, FRANCIS, M.D., Assistant Physician to the East London Hospital for Children; 15, Finsbury square.
- 1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Royal Infirmary, and Lecturer on the Principles and Practice of Medicine, in the Liverpool Royal Infirmary School of Medicine; 69, Bedford street, Liverpool. *Trans.* 3.
- 1878 WATNEY, HERBERT, M.D., Assistant Physician to St. George's Hospital; 1, Wilton crescent, Belgrave square.
- 1837 †WATSON, SIR THOMAS, Bart., M.D., D.C.L., F.R.S., Physician-in-Ordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6.
- 1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. *Trans.* 1.

Elected

- 1879 WATTEVILLE, ARMAND DE, M.B., B.S., 13, Old Cavendish street.
- 1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.
- 1840 WEBB, WILLIAM WOODHAM, M.D.
- 1842 †WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.
- 1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. *Lib. Com. Trans.* 6.
- 1844 †WEGG, WILLIAM, M.D., *Treasurer*, 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-9. *Lib. Com.*
- 1878 WEISS, HUBERT FOVEAUX, 33, Chester terrace, Regent's park.
- 1874 WELLS, HARRY, M.D., British Vice-Consulate, Gualaguay-chu, Entre Rios, Argentine Confederation.
- 1861 WELLS, JOHN SOELBERG, Professor of Ophthalmology in King's College, London, and Ophthalmic Surgeon to King's College Hospital; Surgeon to the Royal London Ophthalmic Hospital; 16, Savile row. C. 1877. *Lib. Com.*
- 1854 WELLS, THOMAS SPENCER, Surgeon-in-Ordinary to H.M.'s Household; Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. C. 1870. *Trans.* 10. *Pro.* 1.
- 1842 †WEST, CHARLES, M.D., Corresponding Member of the Academy of Medicine of Paris; 61, Wimpole street, Cavendish square. C. 1855-6. V.P. 1863. P. 1877-8. *Sci. Com.* 1. *Lib. Com. Trans.* 2.
- 1877 WEST, SAMUEL, M.B., Casualty Physician and Medical Tutor at St. Bartholomew's Hospital; Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 8, Guilford street, Russell square.

Elected

- 1878 WHARTON, HENRY THORNTON, M.A., Surgeon to the Kilburn Dispensary; 39, St. George's road, Kilburn.
- 1828 WHATLEY, JOHN, M.D.
- 1875 WHIPHAM, THOMAS TILLYER, M.B., Physician to, and Lecturer on Clinical Medicine at, St. George's Hospital; 37, Green street, Grosvenor square.
- 1849 WHITE, JOHN.
- 1877 WHITMORE, WILLIAM TICKLE, 7, Arlington street, Piccadilly.
- 1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.
- 1844 †WILDBORE, FREDERIC, 245, Hackney road.
- 1870 *WILKIN, JOHN F., M.D. and M.C., New Beckenham, Kent.
- 1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.
- 1863 WILKS, SAMUEL, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; 77, Grosvenor street, Grosvenor square. *Sci. Com.* 1.
- 1865 WILLETT, ALFRED, Assistant-Surgeon to, and Demonstrator of Practical Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square.
- 1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.
- 1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton [47, Upper Brook street, Grosvenor square]. C. 1849-50. V.P. 1860-1. P. 1873-4. *Sci. Com.* 1. *Trans.* 1.
- 1859 *WILLIAMS, CHARLES, Surgeon to the Norfolk and Norwich Hospital; 9, Prince of Wales road, Norwich.

Elected

- 1866 WILLIAMS, CHARLES THEODORE, M.D., Physician to the Hospital for Consumption, Brompton; 47, Upper Brook street, Grosvenor square. *Trans.* 3.
- 1872 WILLIAMS, JOHN, M.D., Assistant Obstetric Physician to University College Hospital; 28, Harley street, Cavendish square. *Lib. Com.*
- 1859 WILLIAMS, JOSEPH, M.D. Holmhurst, Cambridge park, Twickenham.
- 1868 WILLIAMS, WILLIAM RHYS, M.D., Commissioner in Lunacy; 19, Whitehall place.
- 1839 †WILSON, ERASMUS, F.R.S., late Professor of Dermatology, Royal College of Surgeons of England; 17, Henrietta street, Cavendish square. C. 1877. *Lib. Com. Trans.* 2.
- 1863 WILSON, ROBERT JAMES, F.R.C.P. Edin., 7, Warrior square, St. Leonard's-on-Sea, Sussex.
- 1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.
- 1825 WISE, THOMAS ALEXANDER, M.D., Thornton, Beulah hill, Upper Norwood, Surrey.
- 1879 WOAKES, EDWARD, M.D., 57, Harley street, Cavendish square.
- 1841 WOOD, GEORGE LEIGHTON, 28, Green park, Bath.
- 1851 WOOD, JOHN, F.R.S., Professor of Clinical Surgery in King's College, London, and Senior Surgeon to King's College Hospital; 68, Wimpole street. C. 1867-8. V.P. 1877-8. *Lib. Com. Trans.* 3.
- 1848 †WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8. V.P. 1877-8.
- 1842 WORTHINGTON, WILLIAM COLLINS, London road, Lowestoft, Suffolk. *Trans.* 3.
- 1865 WOTTON, HENRY, M.D., 62, Bedford gardens, Kensington.

Elected

1878 YEO, GERALD F., M.D., M.Ch., Professor of Physiology in King's College, London, and Assistant Surgeon to King's College Hospital; 15, Albemarle street, Piccadilly.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

- 1853 BRODIE, SIR BENJAMIN COLLINS, Bart., M.A., D.C.L.,
F.R.S., Brockham Warren, Reigate.
- 1847 CHADWICK, EDWIN, C.B., Corresponding Member of the
Academy of Moral and Political Sciences of the Institute of France ; Park Cottage, East Sheen.
- 1873 CHRISTISON, SIR ROBERT, Bart., M.D., D.C.L., LL.D.,
Physician-in-Ordinary to H M. the Queen in Scotland ;
40, Moray place, Edinburgh.
- 1868 DARWIN, CHARLES, M.A., F.R.S., Corresponding Member
of the Academies of Sciences of Berlin, Stockholm,
Dresden, &c. ; Down, Bromley, Kent.
- 1857 FARR, WILLIAM, M.D., D.C.L., F.R.S., General Register
Office, Somerset House, and Southlands, Bickley,
Kent.
- 1868 HOOKER, SIR JOSEPH DALTON, M.D., C.B., K.C.S.I.,
D.C.L., LL.D., F.R.S., Member of the Senate of the
University of London, Director of the Royal Botanic
Gardens, Kew ; Corresponding Member of the Academy
of Sciences of the Institute of France ; Royal Gardens,
Kew.
- 1868 HUXLEY, THOMAS HENRY, LL.D., F.R.S., Professor of
Natural History in the Royal School of Mines ;
Secretary to the Royal Society ; Corresponding Member
of the Academies of Sciences of St. Petersburg, Berlin,
Dresden, &c. ; 4, Marlborough place, St. John's wood.

Elected

- 1878 LUBBOCK, SIR JOHN, Bart., F.R.S., High Elms, Bromley, Kent.
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CONTENTS.

	PAGE
List of Officers and Council	v
List of Presidents of the Society	vi
Referees of Papers	vii
Trustees of the Society	viii
Library Committee	viii
List of Fellows	ix
List of Honorary Fellows	lix
List of Resident Fellows, arranged according to Date of Election	lxiii
List of Plates	lxx
Woodcuts	lxxi
Advertisement	lxxiii
Regulations relative to Proceedings	lxxiv

I. Report of the Scientific Committee appointed to investigate the relations of Membranous Croup and Diphtheria	1
— Copies of Circulars of Inquiries	34-7
— Appendix I. Digest of Replies to Queries	39
— II. Report on the History and early Literature of the subject	67
— III. Historical Sketch of Anatomical Distinctions which have been drawn between Croup and Diphtheria	77
History of the Anatomy of Diphtheria in England	77
Ditto in Germany	80
— IV. Report on the Histology of the Laryngeal and Tracheal False Membrane	89
Case of Membranous Laryngitis from Eau de Cologne, by Dr. Whitehead Reid	95
— V. Dr. Dickinson's Tables of Cases, with remarks	101
— VI. Dr. Hilton Fagge's Collection of Cases, with remarks	127
— VII. Dr. Gee's Tables of Cases	149
— VIII. List of Reports to the Medical Officers of the Privy Council and Local Government Board on Outbreaks of Diphtheria, and on subjects related thereto	167

	PAGE
II. A Case of Morphaea. By GEORGE GASKOIN, Surgeon to the British Hospital for Diseases of the Skin . . .	169
III. On the Condition of the Skin in Tinea tonsurans. By FREDERICK TAYLOR, M.D., Assistant Physician to Guy's Hospital; Physician to the Evelina Hospital for Sick Children . . .	177
IV. The Nature of Iodide-of-Potassium Eruption. By GEORGE THIN, M.D. . . .	189
V. Tracheotomy in Membranous Laryngitis: the indications for its adoption, and some special points as regards its after-treatment. By ROBERT WILLIAM PARKER, M.R.C.S., Assistant Surgeon to the East London Children's Hospital; Surgical Registrar to the London Hospital . . .	197
VI. On Three Cases of Distal Ligature of the Carotid and Subclavian Arteries for Aneurism involving the Innominate Artery. By RICHARD BARWELL, F.R.C.S., Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital . . .	217
VII. On Disease of the Mastoid Bone. By W. B. DALBY, M.B. Cantab., F.R.C.S., Aural Surgeon to St. George's Hospital . . .	233
VIII. Observations on the effect of Diet, Rest, Exercise, &c., in Chronic Nephritis. By EDWARD I. SPARKS, M.B. Oxon., F.R.C.S., and J. MITCHELL BRUCE, M.D. Lond., F.R.C.P., Assistant Physician to the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton . . .	243
IX. A Study of the so-called Tendon-Reflex Phenomena. By W. R. GOWERS, M.D., F.R.C.P., Assistant Professor of Clinical Medicine in University College . . .	269
X. On Ophthalmoplegia Externa, or Symmetrical Immobility (partial) of the Eyes, with Ptosis. By JONATHAN HUTCHINSON, F.R.C.S., Senior Surgeon to the London Hospital, Consulting Surgeon to the Royal London Ophthalmic Hospital and Surgeon to the Blackfriars Hospital for Skin Diseases . . .	307
XI. Narrative of a Case of True Leprosy, in which complete recovery has taken place. By JONATHAN HUTCHINSON, F.R.C.S., Senior Surgeon to the London Hospital, &c.	331

	PAGE
XII. On the Diagnosis and Treatment of Ruptured Bladder. By CHRISTOPHER HEATH, F.R.C.S., Holme Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital	335
XIII. Remarks on One Hundred and Fifty Operations for Extraction of Cataract. By CHARLES HIGGENS, F.R.C.S., Ophthalmic Assistant Surgeon, Guy's Hospital; Lecturer on Ophthalmology, Guy's Hospital Medical School	347
XIV. A Case of Secondary Trephining for Traumatic Abscess of the Brain; recovery. By JOHN WHITAKER HULKE, F.R.S., Surgeon to the Middlesex Hospital	367
XV. Cases of Perforating Ulcer of the Foot, with remarks, and an Appendix on the Literature of the Subject. By WILLIAM S. SAVORY, F.R.S., Surgeon to St. Bartholomew's Hospital, and HENRY T. BUTLIN, F.R.C.S., Surgical Registrar to St. Bartholomew's Hospital	373
XVI. On Deligation for Aortic Aneurism of the Right Carotid and Subclavian Arteries, with a new species of Ligature. By RICHARD BARWELL, F.R.C.S., Surgeon to, and Lecturer on Surgery at, Charing Cross Hospital	393
XVII. On the Pathology of Lupus, with special reference to the appearances described as Giant Cells. By GEORGE THIN, M.D.	407
XVIII. Tumour in the Bladder removed by Perineal Incision; complete recovery. By Professor G. MURRAY HUMPHREY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital, Cambridge	421
XIX. The Movements of the Eyelids. By W. R. GOWERS, M.D., F.R.C.P., Assistant Professor of Clinical Medicine in University College	429
XX. Observations on the Ophthalmoscopic Appearances in the Tubercular Meningitis of Children. By GEORGE GARLICK, M.D., late Registrar to the Hospital for Sick Children, Great Ormond Street	441
INDEX	465

LIST OF PLATES.

	PAGE
I. The Skin in <i>Tinea tonsurans</i> . Figs. 1—3. Transverse and Longitudinal Sections of Diseased Hair Follicles. (FREDERICK TAYLOR, M.D.)	188
II. Ditto. Figs. 4, 5. Longitudinal Sections of Hair Follicle and Papilla. (FREDERICK TAYLOR, M.D.)	188
III. Iodide-of-Potassium Eruption. Fig. 1. Section showing second and third described areas. Fig. 2. Part of a Blood-vessel distended and plugged by Granular Coagulated Substance. (GEORGE THIN, M.D.)	196
IV. Perforating Ulcer of the Foot. Figs. 1—4. Transverse Sections of Nerve-fibres. Figs. 5—7. Posterior Tibial Nerve (B—). Figs. 8, 9. Transverse Sections of Nerves (C—). (W. S. SAVORY and H. T. BUTLIN.)	392
V. Ditto. Fig. 1. Transverse Section of Nerve (C—). Figs. 2—4. Sections of Normal Nerves. Figs. 5—8. Sections of Motor and Sensory Nerves. (W. S. SAVORY and H. T. BUTLIN.)	392
VI. Ditto. Coloured Lithograph of the Ulcer. (W. S. SAVORY and H. T. BUTLIN.)	392
VII, VIII. Pathology of Lupus. Figs. 1—11. Sections of Giant Cells, showing their Stages of Formation, &c. (GEORGE THIN, M.D.)	420
IX. The Movements of the Eyelids. Figs. 1—3, Case 1. Partial Paralysis of Left Third Nerve. Figs. 4—6, Case 2. Paralysis of Left Inferior Rectus. (W. R. GOWERS, M.D.)	440

WOODCUTS.

	PAGE
Tracheotomy in Membranous Laryngitis. (R. W. PARKER)—	
Figs. 1, 2. Tube showing Mechanism of the Moveable Collar	202
Figs. 3—5. Sketch of Trachea, with dotted outlines of the ordinary Tube and differently shaped Canula <i>in situ</i>	203-4
Fig. 6. Automatic Retractor	206
Figs. 7, 8. The Croup-bed	207-8
Fig. 9. Plug for Wound in Trachea	213
Distal Ligature of the Carotid and Subclavian Arteries for Aneurisms involving the Innominate Artery (R. BARWELL)—	
Figs. 1—8. Diagrams of the Chest, showing Positions of Pulsating Tumour and Sphygmographic Tracings	220-27
Effect of Diet, Rest, Exercise, &c., in Chronic Nephritis: Chart showing amount of Albumen passed per hour during the Experiments. (E. I. SPARKS, M.B., and J. M. BRUCE, M.D.)	258
Tendon-Reflex Phenomena. (W. R. GOWERS, M.D.)—	
Figs. 1—11. Tracings of Knee Reflex, Ankle Clonus, Toe Clonus, Knee Clonus, Contraction in Gastrocnemius, Reflex Spasm, &c.	276-304
Deligation of Right Carotid and Subclavian Arteries for Aortic Aneurism. (R. BARWELL.)—	
Fig. 1. Arch of the Aorta and Large Branches in section	397
Fig. 2. Heart and Arch of the Aorta and Large Branches	399

1

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REPORT OF THE SCIENTIFIC COMMITTEE

APPOINTED TO INVESTIGATE THE RELATIONS OF

MEMBRANOUS CROUP AND DIPHTHERIA.

IN presenting their report the Committee desire to draw attention to the manner in which the inquiry has been conducted, and the materials upon which their conclusions are based.

At the outset of the investigation it seemed to the Committee desirable that the experience of the Fellows of the Society should, as far as possible, be gathered, and their opinion sought as to the distinctions to be drawn between "diphtheria" and "croup." Their aid was also sought in the view of discovering other reliable sources of information which might be placed at the disposal of the Committee.

With this view a series of questions were framed, a copy of which was sent to each Fellow of the Society, and also to such other members of the profession as were known to take an interest in, or to have had special opportunities for, the investigation of the subject. About 700 copies were issued, and to these 90 replies were received.

A large mass of information was thus collected, and the results of observation of a large number of the most experienced and respected members of the profession in all parts of the world was gathered, and in addition to the formal replies.

to the queries, a number of pamphlets, letters, &c., containing information were sent.

A digest of these replies, and a critical summary of them, are appended to the report.

It was, however, felt by the Committee that the results thus gained, though of great value, would be an insufficient basis for so full a report as the subject demands. In many cases opinions only were given without any statement of facts, and a considerable number of important points involved in the inquiry remained without any reply.

It was, therefore, resolved that a fresh series of questions should be issued, stating certain definite points upon which the Committee desired information, and that a request should be made for the communication of any facts or cases bearing upon these subjects.

Moreover, the experience of the members of the Committee was called in aid, and the data afforded by the large metropolitan hospitals were as far as possible collected. The more important of these have been arranged in a tabular form and are appended to the report. They constitute one of the most important bases of the conclusions arrived at by the Committee, and it is believed that they will form a not unimportant contribution to the known facts upon the subject.

The Committee have also been enabled to make use of the recent reports of inquiries made under the direction of the Medical Officer of the Local Government Board into epidemic and endemic diphtheria, and have derived much information from them; and they have to thank the Medical Officer of the Local Government Board (Dr. Seaton), and the Assistant Medical Officer (Dr. Buchanan), for their courtesy in the matter.

Other materials, such as records of cases, specimens of false membrane, and of the larynx, &c., from cases of membranous exudation have been received from various quarters, and have been examined by members of the Committee deputed for that purpose.

Reports upon the early history of the subject and of inves-

tigations into the morbid anatomy of the diseases in question have also been prepared, and are appended. A report upon the microscopic anatomy of all the cases which have been available is also added.

Although the Committee feel that there is yet much to be investigated before any final conclusion can be reached as to the etiology of membranous exudation in the larynx and trachea, they believe that upon some, at least, of the various questions involved in the subject some light will be thrown by their labours.

OBJECT AND SCOPE OF THE INQUIRY.

The object of the inquiry may be briefly defined as the determination of the disputed question, whether there is such a disease as "idiopathic membranous croup," *i.e.* whether membranous laryngitis exists independently of the diphtheritic poison, and whether, if so, there are any criteria by which it can be distinguished clinically or pathologically.

Two distinct branches of the inquiry are thus opened, *viz.* whether other causes than the process generated by the diphtheritic poison are capable of giving rise to membranous exudation in the larynx and trachea; and whether it is found in practice that such cases occur with sufficient definiteness and frequency to rank as a separate disease. For it is clear that if it can be shown that other causes than the diphtheritic poison are capable of producing membranous exudation in that position, the solely diphtheritic origin of membranous laryngitis must be abandoned. Yet there would remain the questions whether the other causes were not so rare as to be inconsiderable, and whether the morbid product in the two cases was identical.

It may be stated, *in limine*, that there is evidence before the Committee that membranous exudation in the air-passages can be produced by mechanical and chemical irritants apart from the contagious diphtheria; and that a few cases exist in which there is some evidence that membranous laryngitis has followed exposure to cold. It will, however, be seen

that in a large number of cases some other factor must be invoked as a cause apart from or in addition to this, either a morbid systemic condition due to the presence of some constitutional or febrile disorder, or insanitary surroundings, or some other cause not apparent. But there is strong evidence that all the cases thus produced do not fall under the head of contagious diphtheria, and that there are classes of cases distinct from that disease.

Before entering more particularly into these several questions some points may be stated which have embarrassed the investigations of the Committee, and have rendered some of their conclusions only presumptive.

One of the most important of these is the anomalous position which diphtheria holds in the rank of zymotic diseases, and the difficulty of defining precisely what is and what is not diphtheria. It is at present an undecided question whether diphtheria is as distinct and definite a disease as scarlet fever or smallpox, and whether its poison is not readily generated under conditions of foul air and decomposing sewage. The position of diphtheria in this respect is even more undecided than that of enteric fever, and there is still stronger evidence that it may be originated *de novo*, and produce cases which are contagious, and give rise to epidemics. The recent investigations made by the Medical Inspectors of the Local Government Board tend in this direction, and seem to show that diphtheria has a very close relationship with enteric fever in the causes which favour its origin and spread, and its incidence in particular localities either before, after, or together with that fever. The question may therefore be raised whether the sporadic cases of membranous laryngitis which occur apart from the possibility of contagion are not diphtheritic. This latter question could only be decided in the negative by showing that such cases are essentially distinct in their pathology and symptoms from diphtheria, or that they do not possess the property of contagion, or, again, that they are due to the same causes as cases of non-membranous laryngitis; and these points will require further notice.

A large part of the knowledge which we possess on the subject of diphtheria is derived from the study of epidemics, and is open to the objection that in many cases it refers to the more virulent and graver forms of the disease. There can be no question that diphtheria may assume an epidemic form, and that during particular epidemics it may be especially malignant, or may have some particular character, or be followed in a large proportion of cases by certain sequelæ, and that these vary in prevalence in different epidemics.

We know, also, that in large towns diphtheria, like nearly all other specific contagious diseases, is constantly prevalent in a sporadic form, and that it may be endemic in certain localities, even in particular houses and rooms, in which cases occur at long intervals of time, and the disease may thence be spread as isolated cases, or may, from time to time, give rise to a local or general outbreak.

It is clear that these facts to a certain extent embarrass the inquiry in two ways. It may not be justifiable to take the epidemic form of the disease when it affects the larynx as the type with which to compare the sporadic cases of membranous laryngitis, either as regards the symptoms or the sequelæ, seeing that both are evidently largely modified by unknown conditions.

The second difficulty is still more serious, viz. the constant existence of diphtheria in large towns and even in certain centres in country places, and the very great difficulty in tracing the contagion in many cases which are undoubtedly specific as judged by their symptoms and the property of contagion which they possess.

Although the poison of diphtheria is not generally regarded as so contagious as that of most of the specific fevers, there is abundant evidence that within certain limits and under certain conditions it is very highly contagious, and may be conveyed by the most unsuspected channels. (See, for example, a case of conveyance by a pillow in Dr. Thorne's report on the Andover outbreak. Report of Medical Officer of Local Government Board.)

The fact that whilst diphtheria may be very contagious,

especially to certain individuals, yet in some cases manifests no tendency to spread, renders any inference drawn from contagion alone very doubtful. In a very large number of cases the contagion cannot be directly traced; hence the Committee have not felt justified in any given case in excluding the possibility of contagion.

But they have attached some importance to the fact that in a certain class of cases of membranous laryngitis there appears to be much less frequent spread of the disease in a hospital ward than from cases of ordinary recognised diphtheria. (See Dr. Fagge's report in 'Guy's Hosp. Reports, vol. xxii, 1877.)

In the investigation the following points have been taken up in order:

1. The known causes of membranous laryngitis and their relative frequency.

2. The conditions of occurrence as regards association with other diseases, and as to climate, season, and general hygienic conditions.

3. The possible distinctions between classes of cases as regards—

- 1°. General course.

- 2°. Symptoms.

- 3°. Anatomical distribution of the false membrane.

- 4°. Morbid anatomy—local.

- 5°. Morbid anatomy—general.

- 6°. Histological characters.

- 7°. Sequelæ.

1. *Causation.*

It is generally admitted that amongst the causes of membranous exudation in the air-passages the poison of the contagious and occasionally epidemic diphtheria holds a first rank, and that there is a laryngeal form of the disease in which the morbid process is entirely limited to the larynx and trachea.

The point before the Committee only refers to the cases

in which the false membrane is limited, or almost entirely limited, to the larynx and trachea, and it is disputed whether such cases do frequently arise from diphtheritic contagion.

The evidence before the Committee is conclusive as to the fact that in epidemics of diphtheria cases do occur in which the false membrane is thus limited, but it appears from the facts collected that in recorded epidemics such cases are exceptional. Bretonneau stated that he had met with but two instances of this kind, the proportion being one to thirty cases of diphtheria in general. Guersant puts the relative frequency of a primary laryngeal diphtheria at one in twenty cases. In an epidemic which occurred at Auchtergaven, in Perthshire, Dr. Yeats found that among 183 cases of diphtheria there were fifteen in which laryngeal symptoms were present from the commencement, but in which there was no visible affection of the fauces when they were first brought under notice, and in six of them the pharynx remained free throughout the whole progress of the disease ('Edin. Med. Journal,' 1876). The ratio was therefore exactly that which Bretonneau gave so many years before. In a report on an epidemic of diphtheria at Great Coggeshall, Dr. Thorne¹ found that only eight cases out of 180 took this form. Six of these cases were three years of age or under. Nor even in these cases was the possibility of the coexistence of some false membrane in the fauces excluded, for the cases occurred at the outset of the epidemic and their diphtheritic origin was not recognised until later, and no post mortem was made.

From these and other facts it would appear that the frequency of a purely laryngeal form of epidemic diphtheria has been somewhat over-rated.

It need hardly be said that the number of cases in which membranous laryngitis arises from mechanical or chemical irritants, boiling water, and the like is very small, and can hardly enter into statistical consideration. If, therefore, it is shown that in the experience of hospitals a large number of cases are met with in which the false membrane is entirely limited to the air passages, the probability is that some

¹ Report to Medical Officer of Local Government Board, April 11th, 1877.

other condition than contagious diphtheria has been concerned in the production of some of them. They should also if all diphtheritic bear some proportion to the number of grave cases of diphtheria met with.

The tables contributed by Dr. Dickinson, Dr. Hilton Fagge, and Dr. Gee, give some facts which bear upon this point.

Dr. Dickinson's tables give the following results:—In 66 cases of croup and diphtheria—

Membranes in fauces, &c., only	15
In fauces and air-passages (in 10 of which much, in 13 a small quantity only, of false membrane was present in the fauces)	23
In air-passages only	9
Laryngitis, no membranes discovered	19

Dr. Gee's tables:—Out of 76 cases clinically "croupal"—

False membrane was found in the larynx and fauces in	42
In the larynx or trachea only in	21
No membranes discovered	13

Dr. Hilton Fagge's tables:

Membrane in fauces only	22
Marked in the fauces and larynx	25
Slight in fauces and in larynx	9
Larynx only	18
Laryngitis without false membrane discovered	11
Membranous laryngitis directly caused by local injury	7

	Dr. Dickinson.	Dr. Fagge.	Dr. Gee.	Total.
Membranes in fauces only	15	22	—	—
In fauces and larynx	23	34	42	99
Larynx and trachea only	9	18	21	48
Laryngitis, no membranes discovered	19	11	13	43

It would appear from these statistics that there is a very large proportion of cases of membranous exudation in the larynx and trachea as compared with those in which faucial exudation existed alone, and it is noteworthy that there is also a very high proportion of cases of implication of the larynx where there was also faucial exudation, far larger than in any known epidemic of diphtheria.

If we rely upon these statistics alone, we may be led to conclude that a large proportion of cases of exudation in the fauces and air passages together, as well as those of exudation in the air passages alone, are not of diphtheritic origin, as judged by the ratio to the cases of diphtheria free from laryngeal implication. But it is evident that such a conclusion could hardly be warranted by these facts alone, for it must be borne in mind that only the more severe cases, and especially those with laryngeal affection, are brought to hospitals. Too much importance must not therefore be attached to those figures alone, apart from a stricter investigation of the individual cases.

The importance of the ratio of the cases of laryngeal implication is also probably modified by the fact that the greater part of the observations here referred to were made in children's hospitals, and that laryngeal implication appears to occur with greater proportional frequency in children.

Whilst referring to the modified conditions in hospitals, which render a comparison of the statistics of hospital experience with that in families or public institutions somewhat questionable, we may observe that the absence of contagion from hospital cases by no means proves their non-contagious nature. For, although a few cases arose in hospital of those here tabulated, only a very small proportion did so, the number of cases of ordinary pharyngeal diphtheria so arising being inconsiderable. This being so with cases in which the pharynx as well as the larynx was involved, and of which the diphtheritic origin was shown in other ways, it is probable that the number of cases arising by contagion from the purely laryngeal form would be in a still smaller ratio. It is not absolutely proved that contagion is less ready from the purely laryngeal form, but known facts render it probable that it is so. Hence any inference from the proportional infrequency of infection from different classes of cases in which laryngeal membranous exudation is present from whatever supposed cause, must, to some extent, be received with caution.

2. Conditions of occurrence.

As to the predisposing and exciting causes of membranous laryngitis, they may be grouped as determining the affection of the larynx where contagion is present, and as giving rise to it apart from contagion.

Upon these points the Committee have received but little exact information. Many of the causes which have been described as giving rise to membranous laryngitis are known also to favour the affection of the larynx in cases of epidemic diphtheria.

The influences of *age* and of *weather* are two of the most important of these causes which have a twofold action.

Season, weather, and climate.

Some of the evidence given under this head has been mentioned in the analysis of replies to questions (see Appendix), and beyond the fact that variable climate and cold wet weather favour the onset of laryngeal symptoms, no fact of importance was elicited.

Dr. Yeats especially mentions this fact in his report on the epidemic at Auchtergaven.

In the report on the Coggeshall epidemic all the ten cases of laryngeal affection are shown to have occurred in December, November, and January, when also there was much wet and an unusually low temperature. So far as *season* is concerned the most exact facts are those collected by Dr. Dickinson, Dr. Fagge, and Dr. Gee, which are appended.

Laryngeal with faucial exudation :

	Total	Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
Dr. Dickinson	24	2	1	1	1	4	5	1	1	2	0	5	1
Dr. Fagge	35	1	1	4	3	1	1	4	6	5	5	3	1
Dr. Gee	42	3	2	5	4	0	6	4	6	3	3	4	2
Totals	101	6	4	10	8	5	12	9	13	10	8	12	4

RELATIONS OF MEMBRANOUS CROUP AND DIPHThERIA. 11

Laryngeal only :

	Total.	Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
Dr. Dickinson	9	0	4	1	1	0	2	1	0	0	0	0	0
Dr. Fagge .	19	4	0	3	0	1	3	2	1	1	2	1	1
Dr. Gee . .	21	1	1	3	4	2	1	0	0	3	2	4	0
Totals .	49	5	5	7	5	3	6	3	1	4	4	5	1

Laryngeal symptoms ; no false membrane :

Dr. Dickinson	19	1	1	2	1	4	1	0	2	0	0	4	3
Dr. Fagge .	12	2	2	3	1	0	0	1	1	0	2	0	0

Faucial diphtheria :

Dr. Dickinson	15	1	2	0	1	2	2	3	0	0	1	1	2
Dr. Fagge .	(Figures valueless, because of small number of cases, and fact that several arose from one another.)												

Hygienic conditions.

This question is one too wide for discussion here ; the facts with regard to it especially relate to the origination of diphtheria *de novo* or its spread. The only special points with regard to membranous laryngitis as such are given in the analysis of replies to queries. Dr. Dickinson's notes to his table also contain an important observation.

In the Reports to the Medical Officer of the Privy Council and Local Government Board, some further facts are stated, which appear to show that unhygienic conditions which give rise to outbreaks of enteric fever also originate diphtheria.

Age.—There can be no question that membranous laryngitis, whether of proved diphtheritic origin or not, is chiefly a disease of infancy and childhood. This is a fact so completely established by all experience that it is hardly needful to quote facts in its support.

Out of 25 fatal cases of over 180 known attacks of diphtheria in the Great Coggeshall outbreak, only 3 cases were over seven years of age, 3 were seven years, 2 six years old ; all the rest were younger, and of these 17 cases 10 are dis-

tinctly stated to have died with laryngeal symptoms, which were present, so far as can be ascertained, in none of the others.

Of 22 cases of purely faucial diphtheria, collected by Dr. Fagge from the records of Guy's Hospital, only 2 were under seven years of age; in one of these (Case 1) there is great probability that there was membrane in the larynx (the "breathing" was "obstructed"); the other case was not followed till the end, the child being removed from the hospital. Whilst out of 24 of marked faucial and laryngeal exudation, 14 cases were under seven years of age, as was also every one of the 10 cases of marked laryngeal with slight faucial or tonsillar affection. These facts are of especial importance as evidence, because of the very large number of persons over ten years of age in a general hospital.

This very fact, however, renders it necessary, for fair comparison, that all those cases in which the disease was caught in the hospital should be excluded, namely, 7 of the first group and 4 of the second group, all of these being adult patients.

Moreover, others of this first group can hardly be retained, since it is doubtful whether they are really cases of diphtheria at all. The result is that, among eight patients admitted with purely faucial diphtheria, there were only two children, whereas among thirty patients in whom the air-passages as well as the fauces were attacked, there were no less than twenty-four children under seven years of age.

Dr. Dickinson gives 5 cases of non-laryngeal diphtheria under seven years of age, but all the rest were laryngeal alone or combined.¹

Sex.—As to sex, no special influence can in any way be traced.

¹ The majority of cases in the Hospital for Sick Children, Great Ormond Street, are from two to ten years of age; some of the cases, however, occurred in nurses, others at St. George's Hospital.

Association with other diseases.

The relation of contagious diphtheria to epidemics of other diseases is elsewhere discussed (see note to answers to queries); it has no special bearing on the question of membranous laryngitis.

The occurrence of membranous exudation in the fauces and larynx during or after the attack of the disease is one which has been frequently noted, and of which some undoubted facts have been brought before the Committee. They relate chiefly to enteric and scarlet fevers, measles, and whooping-cough.

In all these diseases there occurs, either as a usual or frequent accompaniment, some affection of the throat; in scarlet fever the specific sore throat, in measles and whooping-cough catarrhal inflammation and irritation by cough, in enteric fever, more rarely, tonsillitis and ulcer or abscess of the larynx. The precise relation of the faucial and laryngeal exudation in these cases is a question admitting of much variety of opinion; it may be questioned whether the presence of these throat conditions favours the attack of an unsuspected source of contagion of diphtheria, or whether it is to be regarded as evidence of the origination of true or spurious diphtheria *de novo*. (Other evidence upon this question will be found in the Reports of Dr. Sanderson and Sir W. (then Dr.) Gull to the Medical Officer of the Privy Council, 1858. The association and sequence of scarlet fever and diphtheria are there especially noted. Dr. Gull observed that diphtheria attacks adults especially when they were suffering from some other disease.)

The Committee think it sufficient to call attention to the facts, and to suggest further inquiry with reference to the points—whether these cases are contagious to persons in health, and whether they present, as a rule, the other symptoms of diphtheria, and are followed by its sequelæ; as also whether their occurrence coincides in time with the presence of diphtheria in an epidemic or sporadic form.

It may be pointed out that some of the earliest observations upon the occurrence of diphtheria after measles were made in 1843, by Dr. West, the President of this society.

It had been previously described by Ryland ('Diseases of the Larynx,' 1837, p. 166), who observed it in a number of cases after measles, and in one case after smallpox. Other facts bearing on this subject may be found in the 'Report to the Medical Officer of the Privy Council,' 1858, p. 326.

In several of the cases given in detail in the tables, the membranous exudation involved the fauces as well as the larynx. Dr. Dickinson's table gives two cases accompanying scarlet fever. In many cases of membranous exudation following fevers which have been recorded, and in some of those placed at the disposal of the Committee by Dr. Murchison, the exudation was in the fauces only.

Laryngeal symptoms with no false membrane found :

After measles	.	.	Dr. Dickinson, 2	Dr. Fagge, 1.
„ scarlet fever	.	.	Dr. Fagge,	1.

False membrane in larynx only :

Measles	.	.	Dr. Gee, 1.	
Scarlet fever	.	.	Dr. Gee, 2 (and Sc. F. in house, 2).	Dr. Fagge, 1.
Whooping-cough	.	.	Dr. Gee, 1.	

False membrane in fauces and larynx :

Measles	.	.	Dr. Dickinson, 2.	
Scarlet fever	.	.	Dr. Dickinson, 2.	Dr. Fagge, 1. M. & Sc. F., 1.
Typhoid	.	.	Dr. Gee, 1.	Dr. Murchison.

Membranes in fauces only:

Typhoid	.	.	Dr. Murchison, 2.	Dr. Macpherson, 1.
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There is, however, one point which bears upon the origin of diphtheria *de novo*, and indirectly upon the subject of membranous laryngitis, viz. the occurrence of membranous exudation as a terminal complication of exhausting and septic diseases.

This point has been especially noted by Dr. Wilks in his reply, and he has elsewhere referred to it in more detail.

Origin of membranous laryngitis from a definite exposure to cold.

Notwithstanding urgent and repeated public appeals, no single case of membranous exudation in the larynx resulting from exposure to cold has been furnished to the Committee (though several in which exudation in the pharynx or on the tonsils has been supposed to have resulted from cold have been sent in). The only cases to which reference is made are those collected by the Committee, especially by Dr. Fagge and Dr. Gee. Dr. Fagge has recorded three cases (Cases 58, 59, and 60, loc. cit.) in which this was with some probability ascribed as a cause. Dr. Gee mentions two cases in which cold was stated to be the cause. It is worthy of remark that three cases in which there was also membrane in the fauces were similarly ascribed to cold.

It would appear then that mere exposure to cold and wet is remarkably infrequent as the alleged cause of membranous laryngitis.

Nor have the replies in any of the other points on which inquiry was made contributed anything to our knowledge of the etiology of membranous laryngitis.

3. Course and symptoms in individual cases.

The third part of the inquiry relates to the symptoms in individual cases and in classes of cases, and as to how far they afford any clue to a distinction of membranous laryngitis into different forms.

Very great difficulty has been felt in dealing with the subject on account of the want of any marked symptom or criterion by which provisionally to separate the cases for inquiry. The only one which promised any success is that which has been adopted, viz. the anatomical localization of the false membrane. In a large proportion of cases of diphtheria it is assumed as probable that at some time in the

course of the case there will be some false membrane on some part of the fauces or pharynx, and provisionally this may, in the absence of better distinctions, be accepted. But in practice, there are the greatest difficulties in the application even of this test. In the best marked cases of epidemic diphtheria in infancy the membrane is often in such a position that it is discovered with difficulty during life. In children under five years of age the thorough examination of these parts is almost impossible, large quantities of membrane existing unsuspected in some cases.

Or the membrane in the pharynx may be only a thin pellicular exudation readily detached, and, occurring at the outset only, may be undiscovered during life, and may have altogether disappeared at the time of death, laryngeal exudation then alone existing. This fact diminishes the value of all cases in which a post-mortem examination is not made, and that at a comparatively early period. It will at once be evident that such cases cannot be expected to present either the symptoms or the morbid appearances which are seen in a case where the disease lasts several days, and is attended throughout with grave constitutional disturbance. It is, moreover, allowed that there are *some* cases of true diphtheria in which the air passages are alone affected, and it is clear that whilst they are the only cases which afford a complete type of comparison, it is extremely difficult to select them. It is probable that the presence even of slight affection in the fauces may modify the local and general symptoms, hence the purely laryngeal cases of true diphtheria cannot be expected to present all the symptoms usually attributed to that disease.

The constitutional symptoms must also be greatly modified by any condition which causes rapid asphyxia.

And if we take as the standard of comparison for diphtheria, cases which, while mainly laryngeal, present a small quantity of exudation in the fauces, we are also assuming that all membranous exudation in the fauces, however slight, is due to diphtheria.

It would therefore appear that the only cases which we

can take as a true standard for comparison are those in which the diphtheritic nature is proved by their contagious origin or results, and such cases are necessarily few.

The great fatality which attends membranous laryngitis, as such, renders any inference from the sequelæ of comparatively small value. This is especially the case with regard to the paralysis of the soft palate which is characteristic of diphtheria. It is indeed very doubtful whether this ever occurs even in cases of true diphtheria where the exudation is limited to the air passages; moreover, it occurs in only a small proportion of cases of well-marked faucial diphtheria. These two facts taken together—viz. the very high mortality and the comparative infrequency of paralysis—appear to minimise the value of any conclusions derived from the consideration of this sequela. But it is enough to state that no case of paralysis following membranous laryngitis has been reported to the Committee, or come under their notice.

The mortality in the cases of laryngeal membranous exudation, to which reference has just been made, was in the cases collected

	With faucial.		Without faucial.	
Dr. Dickinson's .	17 of 23	72 p. c.	7 of 9	77·7 p. c.
Dr. Fagge's .	23 of 25	92 p. c.	18 of 18	100 p. c.
Dr. Gee's .	41 of 42	98 p. c.	19 of 21	90·5 p. c.
Total mortality .	81 of 90	90 p. c.	44 of 48	91·6 p. c.

Even in some of the cases of recovery the presence of membrane actually in the larynx and trachea was doubtful. The mortality where laryngeal membrane is found, whether with or without faucial, is 90 per cent.

Mode of onset.

Do the symptoms which attend the onset, the duration of these before the occurrence of croupal symptoms, and the rapidity with which they reach a maximum, afford any ground of distinction?

Suddenness of onset is apparently a characteristic rather of the non-membranous form of laryngitis than of any special class of membranous. It was observed in a certain number, but only in a small proportion of the cases of laryngeal with or without faucial exudation. In Dr. Gee's cases of membranous laryngitis the laryngeal symptoms were observed to set in on the first day in 15 out of 21 of the purely laryngeal cases, and in 16 out of 42 of the laryngeal and faucial, in the remainder of the cases the onset being delayed to the second, third, and so on, to so late as the ninth day.

Fever, malaise, asthenia, or sore throat, were noticed before the laryngeal symptoms in nearly one half of Dr. Dickinson's faucial and laryngeal cases, and about one third of Dr. Gee's. They were present in 23 out of 42 cases of faucial and laryngeal, and in 17 of 21 of purely laryngeal collected by Dr. Gee.

There would thus appear to be a much greater difference between the mode of onset of non-membranous and membranous laryngitis than between any classes of cases of membranous laryngitis. In a very large proportion of cases in which the onset was abrupt, coming on in the course of the night, and the symptoms rapidly reaching their acme, the case, though urgent, did well, and all the evidence led to the belief that there was no false membrane in the larynx.

In Dr. Dickinson's cases abrupt onset in the course of the night was not observed in any single case of pure membranous laryngitis, but occurred in 7 out of 18 cases of non-membranous, and in 3 out of 12 cases of laryngeal with slight faucial exudation.

Symptoms.

Albuminuria.—The presence of albumen in the urine was thus distributed in the cases in which it was observed (these only being mentioned¹).

¹ The number of cases in which it was recorded is small, owing to the difficulty in collecting the urine.

RELATIONS OF MEMBRANOUS CROUP AND DIPHTHERIA. 19

	Faucial.	Faucial and laryngeal.	Laryngeal only.	No. f. m. observed.
Dr. Dickinson .	8 of 15	10 of 13 ¹	2 of 3 ²	
Dr. Gee .	—	22 of 25	7 of 13	0 of 11
Dr. Fagge .	5 of 8	8 of 9	0 of 3	1 of 3
	11 of 18 (61 p.c.)	40 of 47 (85 p.c.)	9 of 19 (47·4 p.c.)	

It appears from the cases collected by the Committee that although albuminuria occurs in both classes of cases, it is relatively much more frequently absent in those of laryngeal exudation only, that it is in much larger quantity where there is marked affection of the fauces, and often precedes laryngeal implication.

In a considerable number of cases of laryngeal form there was no albumen in the urine, in some there was only a trace, and in others it only occurred where there was great obstruction to respiration. If we subtract from these cases all those in which a diphtheritic origin was proved, the proportion of cases of albuminuria would be still smaller. As it is, the ratio is 47·4 per cent. in the laryngeal, 85 per cent. of laryngeal and faucial.

It is evident that while this raises a presumption in favour of the distinction of a non-diphtheritic form of membranous laryngitis it cannot be considered at all decisive, owing to the early fatality and the possibility that the laryngeal affection may modify its presence; and in any given case it will be seen that the criterion of albuminuria would be indecisive.

Moreover it is shown by certain cases, that albumen occurs in the urine in cases of laryngitis, in which no membrane is observed, in one of Dr. Fagge's (Case 85) where no other cause than the dyspnœa could be found.

As to the other general or constitutional symptoms to which some importance has been attached, the Committee have not sufficient facts upon which to base any conclusion.

¹ 4 of 4 where much faucial matter in throat and albumen in large quantity.

6 of 9 " little " " " in 4 in small quantity; one only after tracheotomy.

² One of these was proved diphtheritic (M. Hutson).

Local conditions of the throat.

Swelling of the glands of the neck.—This, so far as the facts at our disposal go, has been far more frequent and well marked where the fauces have been affected, than where only the larynx, and has borne some proportion to the extent and duration of the affection. But in certain of the cases of faucial diphtheria it has been distinctly stated that the glands were not swollen. The glands swollen would probably be different in cases of affection of the larynx, from those where the pharynx was the seat of disease.

(In many cases tracheotomy was performed, and this of course introduces a new element of irritation, and diminishes the value of any conclusion derived from post-mortem examination, the glandular swelling being possibly due to the operation.)

Locality of the false membrane.

The limitation of false membrane to the larynx and trachea is not, as has been pointed out, easily to be determined during life. It is only in those cases examined after death that a certain conclusion can be reached, and even in these there may have been some slight faucial exudation, though none persists after death.

In a very large proportion of the cases in which laryngeal exudation was fatal, more or less false membrane was observed in some part of the fauces before or after death. Great differences are found to exist in the amount of implication of the air passages in different cases.

RELATIONS OF MEMBRANOUS AND NON-MEMBRANOUS
LARYNGITIS.

The large numerical proportion which the cases where membrane in the air passages and fauces, or even in the air passages alone, is to be attributed to some kind of zymotic

influence, bear to those in which it can with any probability be ascribed to cold, together with the uncertainty which forbids us in any case to regard the causation by cold as more than a probability, makes it important to inquire whether there be any mode of distinction between the membranous inflammation and the non-membranous or catarrhal laryngitis, the frequent production of which by exposure to weather does not admit of dispute. The inquiry presents a difficulty in our necessarily less perfect knowledge of the pathological conditions which are followed by recovery than of those which lead to death. In the fatal cases of croup, membrane, as proved by post-mortem examination, is almost always to be found. Among the cases which end in recovery there is rarely any evidence of membrane; but of these our knowledge cannot be such as to enable us to say with certainty that at no time nor to any extent was membrane there. Could it be generally inferred that in a case in the course of which no membrane had ever been seen none had existed, the facts would at once indicate a broad and simple division between membranous croup almost always fatal and non-membranous croup almost always not so.

The facts before the Committee are striking, however they are to be interpreted. Dr. Fagge examined the records of Guy's Hospital without meeting with a single case of croup (using the term in the clinical sense) of which the subject was examined after death without the finding of membrane.¹ One exceptional case afterwards occurred and is mentioned in a foot-note to his tabulation. Dr. Dickinson relates 23 instances of post-mortem examination in cases of inflammatory laryngeal obstruction: membrane was found in 22, the solitary exception being one of œdema of the glottis. And Dr. Gee's search into the records of experience of the other physicians of the Hospital for Sick Children found 60 fatal cases of membranous laryngitis, but not one fatal case of the non-membranous variety.

¹ One such case, which occurred in 1869, has been discovered in the records. The patient was a male child three months old.

Thus, non-membranous croup, whatever be its frequency, is very rarely fatal; it is to be met with, if at all, only, or almost only, among records of recovery.

Cases of croup of which the symptoms closely resemble those of the membranous kind, but in which no membrane is found, and which end in recovery, are numerous; it has to be determined with regard to such whether membrane did not exist or only did not appear. Is there a large class of cases of membranous croup in which no membrane is coughed up or seen *in situ* and which end favorably, or are the majority of such cases of the non-membranous kind?

The facts before the Committee show that even where false membrane was found in the larynx and trachea after death, it was seen during life in but a small minority of the cases in which it was limited to the air passages, and if we exclude the cases in which tracheotomy was performed, the number becomes still smaller. Thus, of Dr. Dickinson's nine cases in which the membrane was thus limited, in only one was its existence shown during life apart from tracheotomy. In any given case, therefore, the fact that no membrane was observed would not negative its existence. And it might be suggested that only in those cases in which the inflammatory process is of sufficient intensity to give rise to false membrane does a fatal result ensue.

It must be allowed that this source of fallacy cannot be entirely excluded, but there are other considerations derived from the study of these groups of cases which indicate essential distinctions between membranous and non-membranous laryngitis.

If we put together all the cases in which false membrane was observed during life or after death, either associated with, or apart from, faucial exudation, we find that those in which it is limited to the larynx and trachea form about one third of the whole. The proportion in Dr. Dickinson's 32 cases was 9 to 23, in Dr. Fagge's 52 cases 18 to 34, in Dr. Gee's 63 cases 21 to 42, so that of a total of 147 cases 48 were affected in the respiratory tract alone.

The enormous fatality of membranous affection of the

larynx of whatever degree is shown by the fact that of those cases in which the membranous nature of the laryngeal affection was rendered probable during life by the discovery of membrane on the tonsils or in the pharynx, recovery took place in only 9 cases of 90, or 10 per cent.

Apart, however, from this head of evidence, and from the consideration of the results of post-mortem examination in cases clinically croupal in character (to which the objection may be raised that the great rarity of the absence of membrane post mortem is due to the fact that the inflammation in the non-fatal cases is not sufficiently intense for its production), we find that in the consideration of this presumed non-membranous class there are some striking distinctions. The differences thus observed are of such a nature as to render it probable that this group of cases is essentially distinct.

If we take these groups, membranous and presumed non-membranous, we find marked distinctions as regards sex, mode of onset, duration, tendency to recurrence and albuminuria.

The distribution between the sexes of non-membranous and membranous croup has its bearing upon this question.

Sex.—The membranous affection of the larynx, like faucial diphtheria, appears to be distributed without sexual preponderance. Dr. Gee's tables, derived from the hospital case-books of Drs. West, Hillier, and Buchanan, show that laryngeal membrane, either thus limited or together with a similar state of the fauces, was found in 28 male subjects, 34 females. Adding to these the cases from Dr. Dickinson's practice, mostly derived also from the Hospital for Sick Children, 15 males, and 17 females, we have a total of 43 males to 51 females thus affected. These numbers at least prove that in the practice of the physicians of the Hospital for Sick Children boys do not suffer from membranous affection of the larynx more frequently than girls.

If there be a class of cases, therefore, in which the sexes are represented in very different proportion the presumption

is that the disorder is of a different kind. Adding together the cases derived as specified from the practice of Drs. West, Hillier, Buchanan, and Dickinson, the tables present 30 males and 12 females as the subjects of croup without evidence of membrane.

Of Dr. Dickinson's 18 cases in which no membrane was seen, 4 are to be reckoned upon less surely than the others, as they were incomplete in consequence of the premature removal of the patient; one in particular, Daniel O'Connell, was taken out in a state of much dyspnoea, having been under treatment for nine days, and might with much likelihood have displayed membrane one way or another had opportunity been afforded.

Putting aside the cases of which the issue is uncertain, there were of 14 cases without visible membrane, 13 recoveries and 1 death, no tracheotomy.

Dr. Gee's collection of cases from the books of the Hospital for Sick Children shows a similar result. Of 13 cases of acute laryngeal inflammation, 'croup' in common phrase, in which no membrane was found, all recovered.

In 63 cases comprised in the same tables in which the larynx was involved and the presence of membrane ascertained there were 3 recoveries to 60 deaths. Tracheotomy was performed in 34 instances. In this comparison an allowance must be made for the more complete knowledge gained of the fatal cases by post-mortem examination than was possible in the others. Thus cases may have been classed as membranous only because they were fatal; since membrane may be evident after death which was not so during life. But even with this drawback the facts are significant.

It will be noted that in the comparison drawn from Dr. Dickinson's abstract this source of error has been avoided by the separation of post-mortem evidence. Dr. Dickinson's tables show that even where the membrane was confined to the larynx it came into view in 3 cases out of 8, while the membrane where less limited and necessarily more often seen, came into view in 19 of 24 cases; in the total of 32 cases in

which membrane was ascertained to exist in the air passages membrane or tonsillar deposit was seen in 22.

	Mem- brane observed during life or after death in	Mem- brane seen during life in	Mem- brane seen only after death in	Mem- brane seen as such in throat	Only white spots or patches on tonsils in	Mem- brane coughed up, in	Mem- brane ex- pelled by tracheo- tomy hole.
2. Membranes con- fined to air pas- sages; 8 cases .	8	3	5	0	1?	1	2
3. Membranes in air passages, with slight affection of throat; 13 cases . . .	13	9	4	4	4	1	4
4. Membranes exten- sively in throat as well as in air passages; 11 cases . . .	11	10	1	10	0	2	2
	32	22	10	14	4	4	8

Of the 22 cases in which membrane was seen during life, 8 ended in recovery, 14 in death. Of the recoveries 6 were after tracheotomy; of the deaths 11 were after tracheotomy, giving a total of 17 out of the 22 in which this operation was resorted to.

Dr. Fagge's collection of cases affords a contrast of the same kind notwithstanding that his results are in one respect very different.

Of 12 cases of croup related by Dr. Fagge in which no membrane was seen the issue was known in 11. Of these recovery took place in 9; death in 2. Tracheotomy was performed in no less than 7 instances, comprising the 2 fatal and 5 of the fortunate cases. In the 2 fatal cases no post-mortem examination was performed, so that their nature may be doubtful. Were they put aside we should have 9 recoveries in as many cases, comprising 5 of tracheotomy; a result unexampled in the history of cases in which membrane has been recognised.

Albuminuria.—In regard to the question of albuminuria in non-membranous croup, the urine in 3 of Dr. Fagge's cases was examined, in one found to be albuminous. Among Dr. Dickinson's were 4 cases in which it was examined; one in which it was albuminous, this exceptional instance being that of the equivocal Daniel O'Connell. In 13 cases of non-membranous laryngitis collected by Dr. Gee the urine was examined in 11 cases, and in all it was free from albumen; in two cases it was not examined. This result is a very striking one. A larger appeal to facts is needed; should the general exemption of non-membranous croup from albuminuria be ascertained, the fact would be both of pathological interest and practical importance.

Mode of onset, &c.—The question as to whether non-membranous croup is more sudden or rapid in onset than the membranous kind is one of much practical interest. Of 19 cases of presumably non-membranous croup, under Dr. Dickinson, 8 began with a sudden seizure in the night, the patient having gone to bed well or only affected with a slight catarrh. Of 21 cases from the same source, in which membrane was known to have existed and was mainly laryngeal (Tables 2 and 3 of Dr. Dickinson's cases), this mode of beginning presented itself in three instances only, showing that though sudden nocturnal seizure is not peculiar to non-membranous croup, it is most frequent with it. Another point of difference is the duration of the illness. Reckoning from the first laryngeal symptom to recovery or death, and taking first the presumed non-membranous class and the cases of recovery, the term of illness varied from 1 day to 44; giving for 14 cases an average of 19 days. Taking for comparison classes 2 and 3 in which the larynx was affected and the pharynx but slightly or not at all, the length of the illness ranged in 5 fortunate cases from 9 days to 35, giving an average of 28 days, showing that the membranous disorder lasts on an average longer, while it is less variable in its duration than the class with which it is contrasted. Looking at the fatal cases, the quick fatality of the membranous class is characteristic; 16 cases of this kind ended fatally at periods

varying from 1 day to 18 days from the first laryngeal symptom, giving an average duration of 4 days. (For further particulars see Dr. Dickinson's tables). The non-membranous class is so seldom fatal that on this point no comparison can be made. A distinction is further to be made in the liability of non-membranous croup to recurrence, while we have no evidence of such a tendency in any form of disease attended with the formation of membrane. Dr. Dickinson's tabulations show that in three subjects of the presumably non-membranous disease attacks were repeated, while with the membranous class there was no instance of the kind save one, in which a child was said to have had "a dozen attacks" which were obviously of a different nature from the contagious diphtheria which at last brought her under notice.

A point of practice frequently noted with regard to the non-membranous class is the occasional urgency of the dyspnoea, suggesting an immediate resort to tracheotomy; and its subsidence, though the operation be withheld, under the influence of time and appropriate treatment. When membrane has been seen there is little to encourage the tactics of delay. Of the 19 cases of membranous laryngitis already referred to as having displayed membrane during life, but 2 recovered without the operation.

DEFINITION OF THE WORD CROUP.

We are told that the word croup, like the word rickets, was in use among the common people, to designate a special kind of disease, long before nosologists borrowed the term. This being so, we know that the name must have been given to appearances of disease seen in the living person, not in the dead body. In other words, croup is a semeiotic term, relating to lesion of function; and, provided we keep within this principle, we may try to improve the current definitions of the word. But if we wish to designate a notion of a kind altogether different, we should not depart from this principle

by wholly perverting the meaning of a word which has long been used in one sense, so as to employ it in another sense; but we should follow the example of Bretonneau, and invent a new term to express the new notion. According to Baglivi's opinion, "That the Moderns should not be opposed to the Ancients, but united with them by a perpetual League, as much as may be; for what can be more indiscreet, than to make them disagree in Words, when they agree upon the Matter?"

To repeat: the word croup relates to lesion of function. The notion, which the term signifies, is not a simple notion, but a complex notion, put together in some such manner as the following:

I. In the first place comes the *Angina laryngea*, the lesion of the laryngeal functions, to wit, the breathing and the voice. The breathing is changed from easy to difficult, and from silent to noisy; the voice becomes hoarse (the exact characters of croupy sounds cannot be conveyed by description, they must be heard to be known); but inasmuch as almost all laryngeal diseases manifest these altered functions, we must distinguish further; and,

II. In the second place, define croup to be an acute disease, sudden in its onset, and swift in its course; but laryngeal disease may be acute and yet not be croup; wherefore we must distinguish further; and,

III. In the third place, add fever to the definition; but acute febrile laryngeal disease is not always croup; and, to complete our definition, we must make more distinctions, namely:

IV. In the fourth place, croup is a primary laryngeal disease; that is to say, it is not secondary to disease in the neighbourhood of the larynx. We do not mean to say that the fauces are wholly free from disease, but that any appearances of inflammation or exudation are slight, and thrown into the shade by the laryngeal lesion.

V. In the fifth place, croup is a disease of childhood. The purpose of this clause in the definition is to debar from the name of croup the acute œdematous laryngitis of adults.

We do not say that adults may not suffer from croup, but that the very uncommon cases of that kind do not admit of absolute diagnosis, excepting by the use of the laryngoscope. In the croup of children the laryngoscope can seldom or never be employed, wherefore we say that croup is a disease of childhood, the other form of acute febrile primary angina laryngea being a disease of adult life.

VI. In the sixth and last place, we will add that croup is spontaneous; that is to say, not due to chemical or mechanical injury of the larynx.

It will be seen at once that, in part, we have argued out Cullen's definition of *cynanche trachealis*:

i. *Respiratione difficili, inspiratione strepente, voce raucâ, tussi clangosâ (i. e. the angina laryngea).*

ii. *Tumore fere nullo in faucibus apparente, deglutitione parum difficili (i. e. primary disease of the larynx).*

iii. *Et febre synochâ (i. e. the fever).* To these clauses we have added three more, in order to make the definition still more precise, namely, croup is an acute disease of childhood, not traumatic. So that our definition runs thus:—Croup is an acute febrile primary spontaneous angina laryngea, occurring for the most part in children.

Divers kinds of Croup.

More than a hundred years ago men began to distinguish two kinds of croup; the one ending almost always in health, the other in death. Hence the names of spasmodic croup as distinguished from inflammatory croup, of false croup from true croup, of stridulous laryngitis from diphtheritic laryngitis, and so on. Whether these two kinds of croup differ in essence or only in degree, whether they correspond with the anatomical species of catarrhal and membranous laryngitis, are points still unsettled. However, this is certain, that we cannot pretend to lay down the grounds of an absolute diagnosis, for the signs are the same in both kinds of disease. But the croup which ends in health may be sometimes

distinguished by a more sudden onset, a more speedy attainment of a high degree of dyspnoea, and less dysphonia.

DEFINITION OF THE WORD DIPHTHERIA.

Bretonneau, who invented the word diphtheria, nowhere gives a formal definition of it. But, by going over his writings, we may pick out for ourselves the meaning he gave to the term, namely:—

Diphtheria is a specific inflammation of mucous membrane or of excoriated skin.

This definition involves two principles, distinct from each other, and distinct also from the principle which we adopted in defining the word croup. The definition of diphtheria is partly anatomical, partly ætiological. We will discuss these parts separately.

I. In the first place, diphtheria is a lesion of structure; it has an anatomical characteristic, namely, inflammation of mucous membrane or of excoriated skin, tending to the formation of concrete exudation upon and within the inflamed tissue. For the words "concrete exudation," Bretonneau sometimes substitutes "false membrane" or "pellicular exudation." But inasmuch as there are many pellicular inflammations which are not diphtheritic, and which anatomy cannot distinguish, Bretonneau had to seek for further aid from ætiology, and,

II. In the second place, to define diphtheria as a specific disease. That is to say, diphtheria is believed to be the result of a peculiar virus acting upon the structures of the body. The virus precedes and underlies the anatomical change. Excepting this result of the virus, very little is known about it; it is sometimes contagious and sometimes epidemic. Wherefore the diagnosis of a diphtheritic pellicular inflammation depends upon two kinds of data; to wit:

1st. Positive, namely, the presence of an epidemic, that is to say, of the diphtheritic virus.

2nd. Negative, namely, by exclusion of other diseases

which are sometimes attended by pellicular inflammations of mucous membrane; for example:

- i. Common membranous or fibrinous angina, as Bretonneau calls it; the "specked throat" of Withering, and the "pharyngeal herpes" of Trousseau; a diagnosis often difficult or even impossible.
- ii. Scarlet fever.
- iii. Dysentery.
- iv. *Oidium albicans*.
- v. Mercurial angina faucium (according to Bretonneau).
- vi. Pellicular inflammation due to the corrosion of poisons.

CONCLUSIONS (SUMMARY OF).

1. Membranous inflammation confined to or chiefly affecting the larynx and trachea may arise from a variety of causes, as follows—

- A. From the diphtheritic contagion.
- B. By means of foul water or foul air, or other agents, such as are commonly concerned in the generation or transmission of zymotic disease (though whether as mere carriers of contagion cannot be determined).
- C. As an accompaniment of measles, scarlatina, or typhoid, being associated with these diseases independently of any ascertainable exposure to the special diphtheritic infection.
- D. It is stated on apparently conclusive evidence, although the Committee have not had an opportunity in any instance of examining the membrane in question, that membranous inflammation of the larynx and trachea may be produced by various accidental causes of irritation, the inhalation of hot water or steam, the contact of acids, the presence of a foreign body in the larynx, and a cut throat.*

* One such case has been communicated since this Report was completed, and will be described in a note to the Appendix upon the Morbid Anatomy and Histology, p. 95.

2. There is evidence in cases which have fallen under the observation of members of the Committee and are mentioned in the tables appended, that membranous affection of the larynx and trachea has shortly followed exposure to cold, but their knowledge of the individual cases is not sufficient to exclude the possible intervention or coexistence of other causes. The majority of cases of croupal symptoms definitely traceable to cold appear to be of the nature of laryngeal catarrh.

3. Membranous inflammation, chiefly of the larynx and trachea, to which the term "membranous croup" would commonly be applied, may be imparted by an influence, epidemic or of other sort, which in other persons has produced pharyngeal diphtheria.

4. And conversely, a person suffering with the membranous affection chiefly of the air passages, such as would commonly be termed membranous croup, may communicate to another a membranous condition limited to the pharynx and tonsils, which will be commonly regarded as diphtheritic.

It is thus seen that the membranous affection of the larynx may arise in connection with common inflammation or with specific disorders of several kinds, the most common of which in this relation is that which produces similar change elsewhere, and is recognised as diphtheria.

In the larger number of cases of membranous affection of the larynx the cause is obscure (*i. e.* in any given case it is difficult to predicate the particular cause in that case).

Among those in which the cause is apparent, common irritation seldom presents itself as the source of the disease, accidental injury is but very infrequently productive of it. But few cases of undoubted origin from exposure to cold are on record. On the other hand, in a very large number of cases infective or zymotic influence is to be traced.

The membrane, even when chiefly laryngeal, is more often than not associated with some extent of a similar change in the pharynx or on the tonsils; and whether we have regard to the construction of the membrane, or to the constitutional state, as evinced by the presence of albumen in the

urine, it is not practicable to show an absolute line of demarcation (save what depends upon the position of the membrane) between the pharyngeal and laryngeal forms of the disease.

The facts before the Committee only warrant them in the view that when it obviously occurs from a zymotic cause or distinct infection and primarily affects the pharynx, constitutional depression is more marked, and albuminuria more often and more largely present, though in both conditions some albumen in the urine is more frequently present than absent.

The most marked division indicated by the facts before the Committee is that between membranous and non-membranous laryngitis.

The Committee suggest that the term *croup* be henceforth used wholly as a clinical definition implying laryngeal obstruction occurring with febrile symptoms in children. Thus croup may be membranous or not membranous, due to diphtheria or not so.

The term *diphtheria* is the anatomical definition of a zymotic disease which may or may not be attended with croup.

The Committee propose that the term membranous laryngitis should be employed in order to the avoidance of confusion whenever the knowledge of the case is such as to allow of its application.

W. HOWSHIP DICKINSON, *Chairman*.

C. HILTON FAGGE.

SAMUEL GEE.

J. F. PAYNE.

H. G. HOWSE.

R. H. SEMPLÉ.

W. S. GREENFIELD, *Secretary*.

The members still serving on the Committee think it right to state that the plan of operations was designed, and much of the earlier work executed, under the auspices of Dr. West as chairman, whose retirement, in consequence of his succeeding to the office of President of the Society, the Committee

have to regret. Since then the chairman has been Dr. Dickinson, and to him also the Committee desire to express their warm thanks.

In the next place the members of the Committee desire to express their sense of the services performed by Dr. Greenfield as secretary, by whom the laborious task of collating the evidence which has been brought together has been mainly performed. If these results should be thought to have any value we are conscious that it must be largely attributed to his unremitting and conscientious work.

Copy of First Circular of Inquiries.

No.

ROYAL MEDICAL AND CHIRURGICAL SOCIETY,
53, BERNERS STREET, OXFORD STREET, W.

SIR,

The Sub-Committee appointed by the Royal Medical and Chirurgical Society to inquire into the relations of "Membranous Croup and Diphtheria" are anxious to obtain the result of the experience and observation of medical practitioners throughout the country on the subject.

They therefore venture to hope that you will, as far as may be in your power, reply to the annexed queries, which have been drawn up with the view of determining the identity or non-identity of these diseases; and that you will further refer the Committee to any practitioners in your neighbourhood who may have had such opportunities for observation as may render their experience on this question of special value.

For the purposes of this inquiry the following definitions are adopted:—

1. Diphtheria is a contagious specific disease, which is accompanied by the formation of false membrane in the pharynx, air-passages, and elsewhere.
2. Croup is a disease accompanied by the formation of false membranes (mainly in the larynx and trachea), the origin of which is in question. No case is to be spoken of as an example of croup in which false membranes were not observed either during life or after death.

We are, Sir, yours, &c.,

CHARLES WEST, M.D., *Chairman.*

W. S. GREENFIELD, M.D., *Acting Secretary of Committee.*

List of Queries.

1. Is your field of observation situated in an urban or a rural district, or partially in both ?
2. What is the nature of the locality as to climate, soil, elevation, proximity to the sea, or to a river ; or as to the drainage or overcrowding of any part of your district ?
3. Have any of these conditions appeared to you to exercise an influence on the prevalence of either form of disease ?
4. At what seasons of the year does either form chiefly prevail ? Do they prevail simultaneously, either sporadically or epidemically ; or may epidemics of the one be distinguished from epidemics of the other ?
5. In connection with what other diseases do they prevail ; either with influenza and bronchitis, on the one hand ; or with angina tonsillaris, or other forms of sore throat, measles, and scarlatina, or other specific disease, on the other hand ?
6. Have you observed cases of croup setting in with catarrhal symptoms, unattended by difficulty of deglutition, or by deposit of false membrane on the fauces, to be associated with albumen in the urine or followed by paralysis ?
7. Have you seen in the same family at the same time, cases of croup in one member and of diphtheria in another ; and have you any evidence to show that membranous croup is contagious, and capable by its contagion of producing diphtheria ?
8. Taking the pathological fact of false membrane limited, or chiefly limited, to the larynx and trachea, what evidence can you adduce as to its origin, on the one hand, in the specific poison of diphtheria, and, on the other, in a definite exposure to cold, or any other cause of ordinary inflammation ?
9. If you believe the two diseases to be identical in nature, on what reasons do you chiefly rely in forming your opinion ?
10. If you have any hospital experience, or experience derived from a large school, will you be pleased to give the Society the benefit of it ?

Copy of Second Circular.

ROYAL MEDICAL AND CHIRURGICAL SOCIETY,
53, BERNERS STREET; May, 1877.

DEAR SIR,

The Committee of the ROYAL MEDICAL AND CHIRURGICAL SOCIETY, engaged in the investigation of the relations of Diphtheria and Croup, are anxious to get some further facts which may enable them to decide whether there exists a non-diphtheritic croup, and by what means it may be distinguished from that due to diphtheritic contagion.

With this object they have drawn up the enclosed scheme for the analysis of records of cases in which the existence of false membrane in the larynx or trachea has been ascertained, during life or after death.

If you have any such records of cases, whether of "croup" or of diphtheritic laryngitis, and will be good enough to give the Committee an account of them, keeping especially in view the points indicated in the appended scheme (these having been selected as bearing on the division of such cases into two classes, and the means by which they may be distinguished), the Committee will be greatly indebted to you.

Should you be prevented by want of time from analyzing the records yourself, the Committee would still be glad of any notes which you may have, and such notes shall be carefully preserved and returned to you.

Yours very truly,

W. H. DICKINSON, M.D., *Chairman.*

W. S. GREENFIELD, M.D., *Secretary.*

I.—ETIOLOGY AND GENERAL HISTORY.

A. PREDISPOSING CAUSES.

- a. i. Season.
- ii. Weather.
- iii. Climate.
- iv. Hygienic Conditions.
- β. Endemic.
- γ. i. Age.
- ii. Sex.
- iii. Hereditary predisposition.
- iv. Constitution.
- v. Previous health.

B. ASSOCIATION WITH OTHER DISEASES.

- | | |
|------------------------|---------------------------|
| (a) As epidemic. | } Whether preceding, con- |
| (b) In the individual. | |
| | current, or following. |

C. EXCITING CAUSES.

- i. Definite exposure to cold.
- ii. Previous attack of other disease.
- iii. Contagion. (Origination apart from definite contagion.)

D. INCUBATION PERIOD.

E. DURATION.

F. PROTECTION FROM FURTHER ATTACK.

G. RELAPSE.

II.—DISEASE—ATTACK OF.

1° MODE OF ONSET.

2° GENERAL CONDITION.

- a. Asthenia (and "adynamic condition").
- β. Condition of Blood.
 - (1) As seen with microscope.
 - (2) Occurrence of ecchymoses, and hæmorrhages.
- γ. Temperature.
- δ. Nervous symptoms.
- ε. Albuminuria (and casts or blood in urine).
 - (1) During attack.
 - (2) After.

3° LOCAL CONDITIONS.

- a. Glandular enlargement, and general swelling in neck.
- β. False membrane.
 - (1) Locality.
 - (2) Spread.
 - (3) Cicatrization as a result.

4° COURSE.

5° PERIOD OF RECOVERY.

6° EFFECTS OF TREATMENT.

(Relative success of Tracheotomy in cases of supposed different forms—*i. e.* whether the presence of one or several constitutional symptoms hinders, on an average, recovery.)

APPENDIX I.

DIGEST OF REPLIES TO QUERIES.

THE replies to the first series of questions issued have been collated and analysed with the following result.

It has been found difficult to arrange them in a complete form, or to introduce all the facts which come out in the statements. Some of the questions were put rather with a view of controlling the results obtained than of eliciting definite information, and where such is the case they are dealt with separately in the consideration of the replies to other questions.

1. Is your field of observation situated in an urban or a rural district, or partially in both ?

On analysing the replies to this question, it is found that by far the larger number of those who have answered are practising in towns, or in districts partly urban and partly rural, only five being in purely rural districts. The numbers are—urban, 27 ; urban and rural combined, 16 ; entirely rural, 5.

This is to be regretted, as there appears to be a tendency to consider that “croup” is more prevalent in rural districts, and in some of the replies it is distinctly stated by those who, at different times, have been engaged in town and country practice, that “croup” is more common in the country. Moreover, it is usually far easier to trace out the causes which give rise to the attack, and to exclude the possibility of contagion in the country than in towns, and climatic conditions are more definitely appreciable, and often exert a greater influence.

2. What is the nature of the locality as to climate, soil, elevation, proximity to the sea, or to a river ; or as to the drainage or overcrowding of any part of your district.

To the first part of this question answers are given of the most various nature. The conditions stated to exist have been

carefully investigated, and so far as they seem to be of value, they have been further mentioned in considering other replies. An attempt was made to classify the several conditions of soil and climate, but the information was not of a sufficiently uniform character to be of service for this purpose. In many cases the conditions of soil and climate are very carefully described, but no facts are stated which in any way connect the diseases with local conditions.

3. Have any of these conditions appeared to you to exercise an influence on the prevalence of either form of disease?

As to the influence of soil, climate, drainage, &c., on the occurrence of croup and diphtheria, thirteen state that they have not observed that these conditions exercise any influence on either. These are—

Dr. CLEMENT DUKES, of Rugby.	Dr. JULIUS POLLOCK, of London.
Dr. LONG FOX, of Clifton.	Mr. W. SMITH, of Clifton.
Mr. FRENCH, of Maida Hill.	Dr. WILKS, of London.
Mr. LEACH, of Heywood.	Dr. WILLIS, of Barnes.
Dr. OSCAR WYSS, of Zurich.	Dr. GRIMSHAW, of Dublin.
Dr. NICHOLLS, of Chelmsford.	Dr. T. STEVENSON, of London.
Dr. O'NEILL, of Lincoln.	

As favouring the occurrence of CROUP the following conditions are mentioned:

Exposed river-side localities, by Dr. HUTTON, of Belgrave Square. Variable climate and moist soil, by Dr. HARRY WELLS, of Gualaguaychu. On gravelly soil, Mr. GASKOIN. In rural districts, Dr. O'CONNOR, of March (diphtheria in urban); Dr. PRICE JONES, of Surbiton (diphtheria in suburban). In crowded neighbourhoods, Dr. WM. SQUIRE, of London.

As favouring DIPHTHERIA.

Proximity to sewage and deficient drainage are mentioned by—

Dr. BARRATT, of Bayswater.	Dr. CHAS. BELL, Edinburgh.
Dr. LANGDON DOWN, London.	Dr. PYE-SMITH, London.
Dr. W. T. GREENE, Old Kent Road.	Dr. W. SQUIRE, London.
Dr. GRIGG, London.	Mr. STRETTON, Kidderminster.
Prof. MACLEAN, Netley.	Dr. DUNCAN, Croydon.

Defective traps.

Dr. BOWLES, Folkestone.	Dr. R. SOUTHEY, London.
Dr. LOWNDS, Egham (late Bombay).	Dr. W. SQUIRE, London.

Impure water supply.

Dr. GRIGG. Dr. HUMBY, St. John's Wood.
Mr. STRETTON, Kidderminster.

Decaying vegetable matter.

Dr. W. YEATS, of Bankfoot.

As favouring both CROUP and DIPHTHERIA.

Low level.—Dr. PAINTER, London.

Overcrowding and bad ventilation.—Dr. RANKE, Munich.

There is thus a large weight of opinion in support of the promotion of diphtheria by insanitary conditions.

4. At what seasons of the year does either form chiefly prevail? Do they prevail simultaneously, either sporadically or epidemically; or may epidemics of the one be distinguished from epidemics of the other?

As to the time of year, the statements need only be classified without the names of those who support them. The numbers appended will give the names, by reference to the table, of those who have replied.

As to diphtheria, the conclusion from these statistics would be that no time of year is especially favorable to its occurrence—it occurs in all alike. But one important fact must be pointed out, viz. that no distinction is drawn as regards the laryngeal form of diphtheria, and whether that form is especially apt to occur at certain seasons.

As to *croup*, the general opinion seems to be that it is especially common in cold and changeable weather.

As to the sporadic occurrence of *croup* the majority state their belief that it is sporadic and not epidemic.

I. SEASON.

DIPHTHERIA.—At all seasons, 4, 19, 79, 373, 436, 534, 567, 641, 643. Spring, 393. Spring and early winter, 366, 622. Early summer, 496. Late summer and autumn, 60, 339, 400. Autumn, 478. Early winter, 311, 614, 627.

CROUP.—Winter, 209, 226, 231, 400, 496, 624, 643. Winter and spring, 28, 60, 79, 162, 567. Spring and autumn, 205, 598. Early summer, 534. Autumn, 593. With east wind, 28, 534. In inclement weather, 291. Rapid changes of weather, 567.*

* Dr. Harry Wells, of Gualeguaychu, Peru, thus describes the conditions which lead to the occurrence of *croup*. "The town (Gualeguaychu) is surrounded by marsh land, and is situated in a hollow on the banks of a small river, Rio

II. *Croup sporadic*, 19, 48, 60, 79, 205, 284, 336, 567, 585, 618, 641. *Croup simultaneous with diphtheria*, 339, 602 (both in epidemics of diphtheria.)

III. *Epidemics distinguishable*.—"No," 435. "Yes." 534. *Neither strictly epidemic*, 643.

No croup seen, 167, 311 (for twenty years), 436, 585 (very rare).

5. In connection with what other diseases do they prevail; either with influenza and bronchitis, on the one hand; or with angina tonsillaris, or other forms of sore throat, measles, and scarlatina, or other specific disease, on the other hand?

With regard to the prevalence of either form of disease in connection with the occurrence of other disease, whether zymotic or due to the conditions of climate or weather, there is considerable difference of opinion.

On the one hand, as regards diphtheria, there is no doubt that this, like other zymotic diseases, is influenced in its outbreaks by general conditions of atmosphere and season, which produce a sort of epidemic influence; and although the exact conditions on which this depends are as yet unknown in their full extent, there can be no question that such conditions do exist, and determine the incidence or severity of nearly all epidemic diseases. The statistics of the Registrar-General, and inquiries by the direction of the Local Government Board, show this clearly enough. Thus measles and scarlet fever are known to tend to prevail together and under similar conditions of climate and season, and even apparently of individual constitution, an attack of the one disease often following that of the other. Nor does the question as to whether diphtheria is a purely zymotic disease, *i.e.* one originated almost entirely, and propagated solely by contagion, to any great degree interfere with this liability to be governed in its outbreaks and epidemics by these general conditions;

Guauguaychu, a tributary of the River Uruguay. Climate temperate, but exceedingly variable, and subject to *rapid* transitions of temperature, 50° to 58° F. in winter, 80° to 88° F. in the shade during summer; latitude 33° south. Drainage none; overcrowding does not exist. There are sudden variations in the condition of atmosphere. The prevalence of a cold south wind and the combined influence of a moist soil and humid atmosphere, invariably produce croup in these regions. Croup is more especially prevalent during winter and spring; however, the rapid changes from heat to cold that continually occur during the summer months (that is to say, a sudden fall in temperature of 20° F.) occasionally causes croup. Diphtheria occurs all the year round. Nevertheless, according to my experience, croup and diphtheria have never occurred simultaneously, either sporadically or epidemically, and the differential diagnosis is easy."

seeing that for erysipelas and enteric fever, which may, in the belief of high authorities, be generated *de novo* by bad hygienic conditions, the same laws are found to hold good. This question of the relation of epidemics of diphtheria with those of scarlet fever and measles, for example, is too wide a one for the present inquiry, and does not immediately affect the problem before the Committee, it must be decided by an analysis of statistics drawn from a much longer period and supported by returns from a far wider area than are at their command. But, as regards individual cases, it is needful to consider one or two points which have been brought forward by some of those who have furnished their experience, before considering their replies in more detail. Firstly, as regards the occurrence of diphtheria in the course of other exhausting or septic disease. Dr. Wilks, in his reply, states that diphtheria may occur as a complication or sequel of typhoid fever, pyæmia, &c.; and Dr. Clement Dukes, of Rugby, makes a similar statement with regard to typhoid. Such cases as those described by Dr. Wilks in which a diphtheritic exudation is found after death in the fauces, pharynx, nares, œsophagus &c., of patients dying from malignant or long-continued exhausting diseases—though rare—are certainly well established. Often the condition is not discovered until post mortem.

The question arises with regard to the cases which may be indistinguishable from diphtheria in *all* their anatomical and clinical characters, how many are the result of contagion, by which they are more liable to be affected. Then arises also the inquiry whether these cases have the property of contagion, and reproduce true diphtheria in the healthy subject coming within the range of their influence. If we allow that such cases are in every respect identical with true diphtheria, we must have recourse to the hypothesis that the source of the patient's infection is "autogenetic" so to speak, that the abnormal conditions of the patient himself, or of secretions, &c., retained and decomposing in the cavities or organs of his body react on the individual in the same manner as similar conditions external to the body may react on a number of individuals. The materials afforded by the replies are insufficient for an analysis of this question.

The late Dr. Laycock in his reply says: "Diphtheria will complicate any epidemic, but more especially those affecting the alimentary canal and air passages. Croup appears to me to be due to constitutional peculiarities, and when these exist the peculiarity may be manifested in any form of disease of the air passages, and more especially in the epidemical as whooping cough, measles, &c."

Another point which it is important to notice, has been well pointed out by Dr. Ransom, of Nottingham, concerning the relation of tonsillitis to diphtheria and scarlet fever. It is

well recognised, both with regard to scarlet fever and diphtheria, that during an epidemic of either disease, cases are very common, in which a sore throat, very slight and perhaps almost unnoticed, and which is almost if not quite indistinguishable from ordinary tonsillitis, is the sole manifestation of the acute disease, yet that such attacks may be followed by the gravest sequelæ, scarlatinal dropsy on the one hand, and diphtheritic paralysis on the other; and, moreover, in the case of scarlet fever, confer protection from a subsequent seizure equally with the most severe attack, though perhaps not in so high a degree. In some cases, more especially in hospitals, or when a family is simultaneously affected with the disease, it is possible both in scarlet fever and in diphtheria to trace the course of such infection. This fact must be kept in view in considering the supposed relation of diphtheria with scarlet fever and with tonsillitis, and it is probable that some of the evidence on this point is invalidated by the fact that this has not been sufficiently clearly understood. But making due allowance for this there is strong evidence for the belief in the prevalence of diphtheria in association with scarlet fever. As regards tonsillitis the case is less clear, seeing that so large a number of mild cases of diphtheria, especially in the adult, completely simulate mild tonsillitis.

The replies under this head must therefore be accepted with some caution, inasmuch as this fact had evidently not been present to the minds of some of those who have made statements with regard to it.

Association of Diphtheria with *tonsillitis*.

Nine state that they have observed diphtheria prevalent with tonsillitis.

28. Dr. BARRATT, of Bayswater: "Proclivity to tonsillitis at the time."

158. Dr. LANGDON DOWN: "I noticed that when diphtheria was prevalent that there was more than usual a tendency to angina tonsillaris."

205. Dr. LONG FOX, of Clifton, says: "In the only epidemic of diphtheria here in the last twenty years there was a great prevalence of angina tonsillaris without scarlatinal rash, and without false membrane."

231. Dr. GREGG (Brit. Hosp. for Ch.), has observed *both* croup and diphtheria.

393. Dr. O'CONNOR, of March, remarked that symptoms of tonsillitis usually occur at the commencement of the attack.

602. Dr. YEATS, of Bankfoot: "Angina tonsillaris was common during the epidemic, and in June, 1876, while diphtheria has been endemic, measles, whooping cough, and catarrhal sore throat have been very prevalent."

496. Dr. REGINALD SOUTHEY.

622. Dr. ALOIS MONTI: "With different forms of sore throat."

629. Mr. STRETTON, of Kidderminster.

641. Mr. G. B. IRVING: "Diphtheria sometimes associated with angina tonsillaris."

643. Dr. G. TENDERINI.

19. Dr. BAKEWELL: "With various forms of sore throat, such as quinsy, inflammatory sore throat, ulcerated sore throat, in all localities observed."

With *Scarlet Fever*.

28. Dr. BARRATT: "In epidemics."

43. Mr. BERRY: "Prevalent with."

79. Sir G. BURROWS: "As a sequela of measles and scarlatina in concurrence with those diseases."

400. Dr. O'NEILL: "Coexisting with scarlet fever and measles in the individual."

435. Dr. RANKE.

598. Dr. C. BELL: "Diphtheria frequently occurs along with scarlatina."

576. Dr. WILKS: "In private practice I have met with diphtheria in connection with scarlatina."

602. Dr. W. YEATS: "Scarlatina prevailed endemically during as well as previous, and subsequent to the diphtheritic and croupy epidemic. Scarlet fever was in some cases followed after an interval by diphtheria and croup, in some cases preceded by, and in some scarlet fever was complicated with diphtheria, in others by croup."

622. Dr. A. MONTI: "Diphtheria has especially followed measles and scarlet fever in epidemics."

624. Dr. W. SQUIRE: "Diphtheria is not an uncommon complication of scarlet fever if it be not an aggravated form of it."

643. Dr. TENDERINI: "With acute exanthemata, roseola, measles, and scarlet fever."

Following *Scarlet Fever* in the individual.

226. Dr. W. T. GREEN.

336. Dr. OSCAR WYSS.

393. Dr. O'CONNOR, Dr. BAKEWELL, Dr. SOUTHERN, Dr. SQUIRE.

618. Dr. STEVENSON doubts whether it is true.

With *Enteric Fever*.—Dr. MACPHERSON.

Association of Diphtheria with *Measles*.

79. Sir G. BURROWS.

336. Dr. O. WYSS observed a great number of cases after an epidemic of measles.

393. Dr. O'CONNOR has observed a large percentage.

400. Dr. O'NEILL.

435. Dr. RANKE.

534. Dr. TREND, following.

576. Dr. WILKS, *doubtful*.

585. Dr. R. WILLIS.

602. Dr. W. YEATS has observed it *with, before, and after*.

19. Dr. BAKEWELL: "With and after epidemics of measles and scarlet fever (as in Hanwell)."

622. Dr. A. MONTI: "Diphtheria has especially followed measles and scarlet fever."

There can be no question, therefore, that diphtheria tends to prevail in epidemics in connection with measles, and is also particularly liable to attack the same individuals especially after the attack of measles.

No relation observed.—158. Dr. L. DOWN.

534. Dr. TREND.

567. Dr. HARRY WELLS.

641. Mr. G. B. IRVING.

RELATION OF CROUP WITH OTHER DISEASES.

The diseases with which croup is observed to be associated, either coincidently as regards conditions tending to produce the two, or as occurring in a person suffering from another disease, may be classed as either produced by similar conditions, or rendering the individual liable to an attack. The number of those who have observed these relations is not large, but their evidence is important, and their testimony very unanimous. On the one hand it is agreed that true croup prevails especially under those conditions which favour the occurrence of catarrhal and inflammatory affections of the respiratory organs, and may occur together with them or successively in the same individual. On the other hand that those zymotic and infectious diseases which specially affect the air passages, as *e.g.* measles and whooping cough, are especially liable to be complicated with croup. Hence, any cause which in the adult would give rise to laryngeal catarrh, may in the child, especially if predisposed, give rise to croup. (It must be assumed that cases of purely *catarrhal* croup are excluded, but even those who recognise most clearly the distinctions between catarrhal and membranous croup allow that in a certain number of cases true membranous exudation occurs.

Influenza, catarrh, bronchitis, and pneumonia.

43. Bronchitis.

60. Catarrh.

- 79. Catarrh.
- 162. Bronchitis.
- 205. Bronchitis.
- 209. Bronchitis.
- 326. Any disease of air passages.
- 336. Croup *ordinarily* alone, in some with pneumonia and bronchitis; *false* croup with bronchitis.
- 393. Bronchitis, pneumonia.
- 400. Influenza, bronchitis, pneumonia.
- 496. Influenza, bronchitis.
- 534. Bronchitis.
- 622. Influenza, bronchitis.
- 624. Influenza, bronchitis, pneumonia.
- 567. Influenza, bronchitis.
- 643. Bronchitis.

Relations of CROUP with—

Measles.—162.

- 226. (This doubtful as in these cases false membrane subsequently in *fauces*).
- 339. Following measles.
- 534. In course of measles.
- 622. Occurring both in the eruptive and desquamative stage of measles. "Bei verschiedenen Epidemien sah ich Croup im Eruptions-stadium oder in Desquamations-stadium der Masern auftreten."
- 624. Dr. SQUIRE.
- 567. *Slight*.

Whooping cough.—326. Dr. LAYCOCK.

624. Dr. W. SQUIRE.

Scarlet fever.—567. *Slight*.

- 598. Dr. C. BELL says: "Croup very seldom with scarlet fever."

Tonsillitis.—624. 231.

Some of the answers to this question (5) are of sufficient value to be given in detail, especially as they introduce some new points as to the association of diphtheria with other diseases.

Dr. WILKS says: "In private practice I have met with diphtheria in connection with scarlatina, and, I think, with measles, and in puerperal women affecting pudenda as well as throat; in hospital practice in typhoid, pneumonia, pyæmia, phthisis, and, I think, other cases. I have seen isolated cases of diphtheritic affection of the throat occur in the hospital previous to the recognition of the disease as a novelty in 1855."

Dr. OSCAR WYSS says: "Diphtheritis laryngis is very often

complicated with diphtheritis pharyngis. Under my care have been in the Hospital for Sick Children (Vienna)—

	1874	1875	Total.
Diphtheritis pharyngis and laryngis, with tracheotomy .	7	10	17
" " without " .	1	4	5
" tonsillarum	1	1	2
" oris	1	0	1
" conjunctivæ ocularis	2	0	2

The greater number of our diphtheritic patients were after scarlatina, or at the same time, but without scarlatinal symptoms; a number of patients had scarlatina some months ago, and after that time diphtheritis. In 1870 there was an epidemic of measles, with a great number of following diphtheritics. The same has been observed this summer, but only in a certain region of the environs of our town (in a poor population). The last year I have seen a certain number of rheumatismus articularis acutus complicated with diphtheria pharyngis."

6. Have you observed cases of croup setting in with catarrhal symptoms, unattended by difficulty of deglutition, or by deposit of false membrane on the fauces, to be associated with albumen in the urine or followed by paralysis?

a. ALBUMEN IN THE URINE.

In reply to this query some state that they have not examined the urine, or have no records of such examination, 28, 43, 79, 226. The latter, however, says that in all his cases of "croup," or nearly all, there was false membrane on the fauces.

Dr. REGINALD SOUTHEY (496) has no records on this point, but all his cases were rapidly fatal.

Dr. WILKS (576) states that he has observed cases which presented the clinical characters in question in which there was albumen in the urine, and they were followed by paralysis. These cases he regards as *diphtheritic*. He adds that the presence of albumen in the urine is unfavorable as regards prognosis, and that in the cases which have had no albumen in the urine, and no sequent paralysis, the proof of presence of false membrane in the larynx and trachea has been generally wanting.

Dr. W. SQUIRE (624) observes that he has seen fugitive albuminuria occurring in children under the influence of trivial and temporary disturbances of health, such as a single night's fever with slight signs of gastric or pulmonary catarrh, and hence would not be surprised to find fugitive albuminuria in the course of an attack of croup, but if albuminuria persisted, he would consider it diphtheria.

Dr. ALOIS MONTI (622) states that he has never found albumen in the urine in cases of sporadic croup such as described.

Dr. G. TENDERINI (643) has not analysed the urine in cases of croup.

Definite observations as to the absence of albumen from the urine in membranous croup.

Dr. BOWLES (60) says: "I have examined several cases of croup as described, and have never found albumen or paralysis, but all my cases have not been so carefully observed. When there has been much obstruction to the breathing, I should not be surprised to find albumen, and if it were present I should by no means consider it diagnostic of diphtheria."

Dr. O'CONNOR (393) says: "I have always paid special attention to the chemical condition of the urine in the two diseases under consideration, and I am strongly inclined to the opinion that in a case of croup occurring in a subject free from strumous taint albumen is not found in the urine."

Dr. OSCAR WYSS (336) denies the existence of albuminuria and paralysis in such cases.

Dr. GRIMSHAW (614) gives two cases of membranous croup, one of which was fatal (and post mortem made), in which there was no albumen in the urine, and in the non-fatal case no paralysis followed.

Dr. BAKEWELL (19) states that he has not observed such cases with albumen in the urine (but that he has seen no case of true croup in the West Indies).

Dr. HARRY WELLS (567) states that "in those cases of croup in which the urine has been examined, the existence of albumen has not been verified, nor has paralysis ever followed an attack of croup in those that have survived."

Those who state that they have found albumen in the urine in true croup are—

Dr. FITZPATRICK (199) describes the case of a child, æt. 2½ years, with symptoms sudden in their onset, and resembling those of "acute inflammatory croup." Fauces not visibly affected. Albumen was found in the urine, and tracheotomy being performed false membrane was withdrawn from the wound. Dr. Fitzpatrick gives details which show that he considers the case diphtheritic as to its etiology. The sequel of the case is not given.

Dr. LONG FOX (205) states that he has seen albumen in the urine temporarily once only in croup.

Dr. W. YEATS (602) states that he has seen it in several cases, that in many of these false membrane appeared subsequently on the fauces, but not in all. All his observations were, however, made during an epidemic of diphtheria, and therefore are not cases of non-diphtheritic croup.

b. OCCURRENCE OF PARALYSIS AS A SEQUEL OF CROUP.

The great majority state that they have never seen true membranous croup followed by paralysis.

- | | |
|--|------------------------|
| 28. Dr. BARRATT. | 496. Dr. SOUTHEY. |
| 79. Sir GEO. BURROWS. | 567. Dr. H. WELLS. |
| 284. Dr. HUMBY. | 614. Dr. GRIMSHAW. |
| 336. Dr. OSCAR WYSS. | 624. Dr. W. SQUIRE. |
| 393. Dr. O'CONNOR (except in case of hereditary syphilis). | 641. Dr. IRVING. |
| 467. Dr. SANSOM. | 643. Dr. G. TENDERINI. |
| | 622. Dr. ALOIS MONTI. |

The only statements of the existence of paralysis following croup are as follows:

Dr. GREENE (226) says: "I have observed several cases of croup quite without difficulty of deglutition to the very last, which were ushered in with catarrhal symptoms. False membranes on the fauces have been so generally present that I cannot recal one instance to the contrary. One case was followed by temporary paralysis of the left side." (From which it is clear, firstly, that all the cases were probably diphtheritic, and that the only paralysis observed was a slight hemiplegia, probably due to thrombosis or embolism.)

Dr. RANKE, of Munich (436), says that he has observed croup followed "by paralysis of the larynx, so that for a time part of everything swallowed entered the windpipe and was coughed up again." (But from other parts of his replies it appears that he speaks of diphtheritic croup. He says: "I should think that of the cases of croup I have seen, at least nine tenths occurred after diphtheria." This suggests a temporary paresis due to the direct effects of inflammation.

Dr. YEATS (602) also mentions cases, but all were a part of an epidemic of diphtheria.

7. Have you seen in the same family at the same time, cases of croup in one member and of diphtheria in another; and have you any evidence to show that membranous croup is contagious, and capable by its contagion of producing diphtheria?

As regards the question of the possibility of *contagion* of membranous croup, by far the larger number of replies strongly negative the idea of such a possibility: Out of 28 who have answered this question, 25 answer in the negative, only 3 affirming the possibility of contagion.

Those who reply in the negative are—28, 43, 60, 79, 162, 205,

226, 231, 336, 339, 373, 388, 400, 420, 436, 496, 534, 585, 615, 618, 629, 622, 467, 567, 614, 643.

Two qualifications must be made.

Dr. RANSOM, of Nottingham (436), excepts cases in which diphtheria attacked the trachea and larynx so early and so severely as to make the pharyngeal affection relatively unimportant, which statement is only a confirmation of the same fact. But Dr. Ransom says that he has seen no cases of true croup in his own practice. All the others speak of it as personally known to them.

Dr. WILLIS, of Barnes (585), has seen no reason to believe in the contagiousness of diphtheria.

Those who make statements as to contagion in croup are—

641. One case stated.

Dr. TREND, of Southampton (534), makes an important statement on this question. "I have noticed in large families and boarding schools that sometimes membranous croup seems contagious, as more than one will take it, but this I have attributed to their being exposed to the same influence. I have never seen croup in one produce diphtheria in another. In the same family I have seen children with measles get croup, followed by diphtheria, while other members of the family had scarlatina."

Dr. LANGDON DOWN (158) also says: "I have known at the London Hospital two children with membranous croup brought from the same house, *but I have never known diphtheria to be produced in hospital from croup.*"

In reply to the inquiry as to the occurrence of croup with diphtheria in the same family, 23 state that they have never known them occur together.

Dr. WILKS says: "I have seen in the same family instances of the ordinary diphtheritic affection of the throat, and at the same time the laryngeal or croupous form. I have seen instances where the affection was confined to the fauces in one member of the family, where it extended to the larynx in another, and where it has commenced in croup in a third, showing how all these affections may be one and the same. In the last case, if it had occurred alone, it would probably have been regarded as the old-fashioned croup, being in no way distinguishable. I have no notes of any case where the disease began in the larynx and was regarded as simple croup, and subsequently other members of the family had the pharyngeal form, but I think I have seen instances of it."

8. Taking the pathological fact of false membrane limited, or chiefly limited, to the larynx and trachea, what evidence can you adduce as to its origin, on the one hand, in the specific poison of diphtheria, and, on the other, in a definite

exposure to cold, or any other cause of ordinary inflammation?

The replies to this question are few in number and not very decisive.

The question was framed partly with the object of deciding whether the *limitation* of the false membrane to the larynx and trachea was a point of importance in deciding whether it has its origin in a specific poison or no.

It is therefore important to ascertain whether in cases of undoubtedly diphtheritic origin such limitation of the false membrane is ever observed.

Dr. RANKE (435) mentions the case of a child in whom tracheotomy was performed for "membranous croup," having at the time no false membrane on the fauces. Three days later false membrane appeared on the fauces. Other members of the same family had at the time, or afterwards, true diphtheria of the fauces.

Dr. YEATS (629), in his pamphlet and reply, gives cases bearing on this point.

Dr. B. WOODMAN gives cases of laryngeal affection occurring alone in the case of an infant aged eighteen months and in another aged two months, and also others where other members of families to whom they belonged had diphtheria. He states that it was limited to air-passages.

In opposition to this view, that the false membrane in diphtheria may be limited to the larynx and trachea, are the statements of Sir Geo. Burrows and Dr. Long Fox.

Sir GEO. BURROWS (79) says: "I have never seen a case of croup arising in connection with diphtheria," and expresses his belief that when the poison of diphtheria is in operation it appears to affect several persons simultaneously or in rapid succession.

Dr. LONG FOX (205) states that he has never seen such limitation to the larynx and trachea in diphtheria, although he believes that in certain epidemics it may be so limited.

Dr. HARRY WELLS (567) draws attention to the "marked tendency that diphtheritic exudation affecting the fauces, tonsils, pharynx, and nasal mucous membrane, has to extend itself to the larynx and trachea, and even to the bronchial tubes, leaving slight traces of its original site."

On the second point, viz. whether such membranous exudation may be produced by exposure to "cold or other cause of ordinary inflammation" there are several very definite statements, some ascribing the origination to cold only, others to the influence of cold and other exciting causes of catarrh upon those suffering from the effects of other diseases, especially measles.

Sir GEO. BURROWS (79) says that he has frequently seen croup arise from a definite exposure to cold.

Dr. LONG FOX says: "Very many times in my practice has croup been referred to damp and cold. When my children were younger the imperfect drying of a washed night-nursery floor has on several occasions seemed the origin of croup." (It is difficult to believe that Dr. Fox is speaking of membranous croup only, yet he has so very clear views on the differences of diphtheria and croup, both as to characters of false membrane, &c., that if not so, it must have been an inadvertent statement.)

Dr. GRIGG expresses a belief that croup always arises from wet and cold.

Dr. O'NEILL, of Lincoln, states that he has generally made out satisfactorily to himself that the occurrence of false membrane limited to the trachea was due to exposure to cold or other causes of ordinary inflammation. He adds that he has seen an hereditary tendency to croup in families.

Dr. RANKE, of Munich, says that he has seen, in addition to the cases of diphtheritic croup, also cases occurring spontaneously and sporadically, where no other cause for the disease could be assigned than exposure to cold.

Dr. W. SMITH, of Clifton, expresses a belief in the occurrence from exposure to cold.

Dr. TREND, of Southampton, says: "To my knowledge I have never seen croup arise from the specific poison of diphtheria, but, as stated above, I have seen it often from that of measles. On the other hand, I have frequently traced it to exposure to cold, and seen in the same family some children from the same exciting cause with croup, and others with bronchitis. I have noticed that the children who get croup have generally disordered digestion and loaded bowels."

Dr. SQUIRE, in reply to this question, does not state whether he has seen false membrane limited to the trachea and larynx in diphtheria, but he says that mere exposure to cold will seldom of itself produce this result of inflammation of the larynx and trachea. It may require the presence of the specific fever of measles or of influenza, or the lowered health state produced by them, or by hooping-cough, or by overcrowding, to convert ordinary catarrhal inflammation into a deep-seated low form of inflammation. Individual susceptibility or idiosyncrasy is also of some account in the process." At the same time Dr. Squire agrees in the view that a great proportion of the cases of membranous exudation in the larynx and trachea occur in the course of diphtheria.

Mr. STRETTON says: "I never attended a case of croup without clear evidence as to cause in careless exposure or vile dietary errors."

Dr. SANSOM says: "I have never seen evidence which is

contrary to the conclusion that false membrane limited to the *trachea* takes its origin from exposure to cold or causes of ordinary inflammation."

Dr. MONTI states that he never saw croup of the larynx and trachea from diphtheria without other false membranes elsewhere.

Dr. BAKEWELL, in answering this question, makes statements of importance as to the non-existence of diphtheria in Trinidad before 1869, but does not state that croup existed either. Has not seen croup since he left England in 1866. Saw much croup, never diphtheria before 1857.

Dr. WOODMAN, on the contrary, gives cases of "croup" from diphtheritic contagion, but states that though he has seen false membrane from scalds, poisons, &c., when exposure to cold has produced laryngeal symptoms, he has never seen any false membrane. (This bears also on the question of production of false membrane by scalds and other severe irritants.)

The following statements by Dr. SANSOM bear on the subjects treated both in Questions 7 and 8, and may be here introduced.

"Seeing, then, that (a) cases having the clinical characters of true croup (and confirmed by the results of autopsies) go through their course without the *demonstrable* presence of false membranes, that (b) post-mortem examinations whereby such membranes are rendered evident occur in only a small minority of observations, and that (c) cases of moderate severity may recover in which the mucus expelled from the trachea exhibits no fibrillar or membraniform arrangement, I feel that I must protest against the definition which renders the demonstration of false membrane during life or after death a *sine quâ non* for the diagnosis of true croup. This, in my opinion, is equivalent to a *petitio principii*.

"It is possible, therefore, that my observations may be rejected by the Committee as not fulfilling the conditions of the inquiry, but I send them that they may be taken for what they are worth.

"In the cases I have designated 'true croup' the onset of the characteristic symptoms has been preceded by a malaise of not more than two or three days—the subjects have been in nearly every instance presumably healthy—*i. e.* presenting no signs of cachexia, but usually rotund and florid. The earliest stages have been those of catarrh, with uneasiness referred to the throat; there has always been some pyrexia; the dyspnoea, though subject to exacerbation, has been continuous and progressive.

"DIAGNOSIS A, from spurious croup, *i. e.* spasm of the adductors of the vocal cords. In the majority of cases this has been very easy. The conditions in the latter have been notably those of disturbance of the digestive organs, a proneness to intestinal worms, and, in a few cases, syphilis. The dyspnoea exhibits distinct remissions, either without obvious cause or in response to measures taken

to provoke enuresis, purgation, or inhalation of anæsthetics. In croup, on the other hand, though there are variations of intensity, provoked by cough and by conditions of irritability, there is, nevertheless, a steady increase in the dyspnœa, an advancing cyanosis, and impairment of peripheral circulation. In a few cases, however, the diagnosis may be difficult. In one case under my care there was no obvious remission of the dyspnœa, but in this instance the child was cachectic, and probably syphilitic. At the post-mortem examination no other sign was discovered than violet colouration (venous congestion) of larynx and trachea. It was probably an example of paralysis of the abductors of the vocal cords, but the child was too young to allow of a laryngoscopic examination being made. Where the vocal cords cannot be observed the diagnosis from true croup can be formed by the absence of pyrexia, and by the general dyscrasia."

"B, from diphtheria. I accept the definition of the Committee with this comment: That I have always observed the diphtheritic false membrane to originate upon the mucous surface of the tonsils, the pharynx, the uvula and palate, or the buccal cavity. The diagnosis from croup is afforded by—(a) the profound adynamia, (b) the secondary inflammation and enlargement of the neighbouring lymphatic glands, (c) the appearance of diphtheritic false membrane upon any wounded surfaces, (d) the infiltration of tissues contiguous to this membrane, with sloughing of the soft parts, (e) the occurrence of various nerve-paralyses at a later period of the disease, paralyses which I have never seen in the cases which have recovered from imminent death from croup attended with abundant tracheal false membrane, (f) the proved contagiousness of diphtheria; not a single case of contagion has been observed by me in true croup."

9. If you believe the two diseases to be identical in nature, on what reasons do you chiefly rely in forming your opinion?

(The larger number of the replies to this question are, strictly speaking, irrelevant, for they consist, either of a statement that the writer does *not* believe them to be identical, or of reasons for the belief that they are not so).

Only a few state their belief that the two diseases are identical, but some argue in favour of this view, though not committing themselves to a definite opinion.

Dr. RANSOM says that he suspends judgment because he has only found after death in croup a little ulcer or signs of slight inflammation without false membrane, and of such cases he has seen very few. Of cases of true diphtheria, which might have been called croup, he has seen many. He inclines to the view that nearly all cases of membranous croup are instances of a mistaken diagnosis.

(Dr. SHEARMAN, of Rotherham, believes in their identity, but says that he was convinced by Dr. Geo. Johnson's paper, not by his own experience.)

Dr. RANKE, of Munich, believes that nine tenths of the cases of croup are due to the diphtheritic poison, but he believes that it may arise from other causes, though his own experience does not afford him any proof of it.

Dr. YEATS, of Bankfoot, believes them to be identical, but his experience is mainly derived from an epidemic of diphtheria and from scattered cases obviously closely related to the epidemic. The statements which he makes and the arguments employed evidently refer to croup of undoubtedly diphtheritic origin, and therefore hardly bear on the question at issue.

Prof. STORCK makes a positive statement that they are identical, but his statements are open to a similar objection.

Dr. BATHURST WOODMAN, whose experience seems to have extended over a wide area, makes some important statements, but their value is diminished by the fact that all cases in which the false membrane is limited (or apparently so) to the larynx and trachea, even where there is definite evidence that the disease is of diphtheritic origin, are spoken of by him as "croup," and this must be considered in judging of the statements. Thus, he gives three cases of croup, one of a girl, aged eighteen months, suffering from laryngeal diphtheria, and the brother, four years old, at the same time with false membrane on the mouth and pharynx. In another case, two children brought to the London Hospital, one with diphtheria, the other with "croup," and another died of "croup" the ages (are not stated). Another case, in which a child, three years old, went home to Torquay with paralysis of the pharynx and a patch of false membrane on the tonsil, and slept with an infant brother, two years of age, who caught "croup" and died. These cases show only what was already recognised, viz. the existence of membranous laryngitis due to diphtheria. On the other hand, Dr. Woodman makes the important statement that "he has seen false membrane from scalds, poisons, &c." He adds that when exposure to cold has produced laryngeal symptoms he had never seen any false membrane. Dr. Woodman's experience includes about fifty cases of diphtheria and croup seen in general and hospital practice. Dr. Woodman also refers to the returns of the Registrar-General, as showing how diphtheria has replaced croup.

These are all the more important arguments advanced in favour of the view of the "identity" of diphtheria and croup. Other replies sometimes repeat one or two of these, but need not be given in detail.

On the other hand, there is a large numerical majority of those who express a belief that membranous croup is not in all cases to be ascribed to diphtheria. The greater number have simply

recorded their opinion to this effect, but some few have given the ground on which it is based. As these are to a great extent similar on some points in particular, these points may first be considered together, and then any special grounds of distinction stated.

1. The general condition of the patient. Great stress is laid on this by several observers. It is stated that asphyxia is the sole or most prominent symptom in croup, whilst asthenia is usually combined with the asphyxia in diphtheria. Connected with this is the mode of onset before the laryngeal symptoms have become prominent. It is also held by most of those who state these distinctions, that the treatment which is successful in the one class is not so in the other, that the one requires an antiphlogistic, the other a stimulant treatment.

The chief names of those who especially insist on this point are:—Dr. Barratt, Dr. Duckworth, Dr. Long Fox, Mr. Gaskoin, Mr. Humby, Mr. Leach, Dr. O'Connor, Dr. Southey, Dr. Maclean, Dr. Alois Monti, Dr. Pye-Smith, Dr. Sansom, Dr. Wells, Mr. Irving, Dr. Tenderini. All these express and others imply that there are very marked distinctions in this respect.

2. The infectious character of "croup" when of diphtheritic origin; the comparative absence of infection in other cases.

This point is especially referred to by several, but no further facts are adduced in support of it than those already given. Dr. Barratt, Dr. Duckworth, Mr. Leach, Dr. Maclean, and Dr. O'Connor, especially lay stress upon this. (But it is only fair to say that they appear to speak of diphtheria as a whole, and not merely of the contagiousness of the special class of cases of purely laryngeal diphtheria as contrasted with the presumed non-diphtheritic class.)

3. The different nature of the exudation in the air-passages.

Dr. Long Fox and Dr. Lionel Beale especially refer to this as distinctive, but they give no precise information as to the exact nature of the difference.

4. The locality of the false membrane.

The principal questions which arise under this head are—*a.* Does false membrane ever occur in the fauces in cases of non-diphtheritic croup? *b.* Are there cases of diphtheria in which false membrane is entirely limited to the air-passages? *c.* Is there a greater tendency for the false membrane to spread to the bronchi in diphtheritic than in non-diphtheritic croup?

Dr. Duckworth, Dr. Long Fox, Dr. Greene, Mr. Hutton, Dr. Laycock, and Dr. Southey, all insist upon the limitation of the false membrane to the larynx, trachea, and bronchi, as peculiar to croup, but they do not state certainly that such limitation is impossible in diphtheria, nor do they give any precise information upon it.

5. As to the age at which "croup" occurs the statements are few. One answer states that croup does not occur after five years of age; another that it spares early infancy.

6. Glandular swelling in the neck is one point especially insisted on by Dr. Duckworth, Dr. Pye-Smith, and Dr. Wells. They state that it is universal in diphtheria, but absent or nearly so in croup. (They do not, however, say whether this may not depend upon the part affected.)

Such are some of the principal grounds on which the distinction is based. Some few remarks upon other points which may aid in the consideration of the subject may be added.

Dr. PRICE JONES lays especial stress upon the difference of climate and locality in relation to the two forms, contrasting his experience in practice in different localities. In Wales he found the laryngo-tracheal affection common, whilst in Surbiton he has observed the throat affection to prevail especially. (It is not stated whether this is to be regarded only as evidence that climate influences the part affected.)

Dr. LAYCOCK says a false membrane is only a limited part of the anatomy of diphtheria, whereas it is the chief anatomical characteristic of croup.

Dr. O'CONNOR (whose experience appears to have been exceptionally great) draws especial attention to the fact of the occurrence of diphtheria in the children of the poor, especially those in a low condition, whilst the cases of "croup" in his experience were chiefly among the well-to-do.

Dr. WELLS says: "My experience hitherto does not lead me to conclude that these diseases are identical in nature. I would classify croup as an inflammatory affection of the trachea, below the glottis, with exudation of albuminous material, accompanied by spasmodic action of laryngeal muscles, attacking children from the eighth month up to the seventh year; whilst diphtheria is pre-eminently a specific disease, rapidly producing profound alterations in the blood, occurring in all climates and all seasons, and, in contra-distinction to croup, affects subjects of *all* ages, and is exceedingly contagious.

Dr. ALOIS MONTI says: "From a clinical stand-point it cannot be certainly decided whether the two diseases are identical. If one also considers that the two are only different degrees of the same affection, the difference of the clinical appearance must be treated, the grounds of which are: Croup is a local affection of the larynx and trachea, while diphtheria is a general infectious disease, with localization in the pharynx, larynx, trachea, &c. Croup is, like every inflammation, induced by cold (*erkältung*); diphtheria by a specific contagium. Croup is not contagious; the power of contagion of diphtheria is allowed. Croup runs its course only as a local disease, and kills only as a consequence of stenosis, diphtheria runs the course of a general infectious disease that

kills rather as a consequence of blood poisoning than by sequel of stenosis. Croup never leads to paralysis, nor is there ever disease of the kidneys during it; in diphtheria this is highly prevalent.

"The lymphatic glands are either not swollen in croup, or only in a slight degree. In diphtheria the lymphatic glands are constantly swollen, and it depends upon the stage of the disease whether this results in virulent inflammatory buboes."

10. If you have any hospital experience, or experience derived from a large school, will you be pleased to give the Society the benefit of it?

In the replies relating to school and hospital experience, some important facts are stated.

Dr. LANGDON DOWN, in an experience of ten years at Earlswood Asylum, says that he has never seen a case of membranous croup among the inmates, though laryngismus stridulus frequently. Diphtheria in mild forms epidemically two or three times.

Dr. CLEMENT DUKES, during five and a half years of experience at Rugby, and as physician to Rugby School, has never seen a case of croup, and only one of diphtheria.

Dr. LONG FOX, as the result of nineteen and a half years of experience as physician to the Bristol Infirmary, states that he has seen a large number of fatal cases of diphtheria and croup, and autopsies. He draws marked distinctions between the two (for which see elsewhere), saying that he has never seen a case of croup where the false membrane was above the glottis, nor ever a case of diphtheria where it was not above.

Mr. FRENCH, as the result of forty years' experience as medical officer of St. James's Parish, with charge of the workhouse and supervision of the parish schools at Brentford and Wandsworth Common, has seen no marked epidemic of diphtheria, but cases of croup in infants occasionally, which usually yielded to leeches and antimony.

Dr. LOWNDS has seen no cases of membranous croup, but cases which, he believes, would have gone on to membranous if left alone. Ipecacuanha emetics caused vomiting of a pultaceous mass, and no further symptoms occurred. The convalescence was "quite different" from that in tracheal diphtheria.

Dr. NORMAN MOORE makes the following statement: "In St. Bartholomew's Hospital I have often seen croup, and rarely diphtheria, 1869—76."

But Dr. REGINALD SOUTHEY, drawing his experience from the same hospital and from dispensary practice, says "that at St. Bartholomew's he has had, under his care many cases of croup

and of diphtheria. The cases of croup die, without a single exception, with symptoms of suffocation. A certain proportion of the cases of diphtheria recover, these last never fail to present albumen in their urine, and are often, but not invariably, succeeded by some local and in some cases general spinal paralysis.

"The cases of croup occur sporadically, one member of a family only is affected at a time, and the disease does not spread in the neighbourhood."

"The cases of diphtheria have appeared to be highly communicable; wherever I have seen one case in a family, other members of the same family have become affected within a short space of time (three days to six weeks). Last year I had four cases of diphtheria from one house in St. Pancras Parish, two children, father and mother, all died with symptoms of well-marked blood poisoning and local throat and tonsillar deposition. They died when they were apparently better and beginning to convalesce quite suddenly by asthenia or sudden heart arrest."

Dr. O'CONNOR says that croup may be membranous or not, hence does not distinguish.

Dr. O'NEILL, of Lincoln, gives some facts indicating that (as is commonly believed) croup does not occur over ten years of age, and that "croup" is not contagious, whilst diphtheria is highly so.

In a girl's school (ages from ten to eighteen) he never saw a case of croup in some years, but a case of diphtheria which was sent him infected *three* adults with diphtheria. In contrast with these, he had two cases, aged three and five, of croup in different families, one in Lincoln, the other in the country, at the same time; both were in families of children, but although they could not be kept separate from the others, and one died (the other recovered), no one else took the disease. This fact alone would be of great value in showing that either croup is distinct, or that there is far less liability to spread contagion when the larynx and trachea only are affected.

Dr. A. J. POLLOCK during a period of twelve years as physician to the Foundling Hospital, states that he has never seen a case of croup, and only three of diphtheria, which occurred at once, and two were fatal. In his other hospital experience at King's, and Charing Cross, he has seen no diphtheria and only about six of croup.

Dr. RANSOME, of Nottingham states that, during twenty years as physician to the General Hospital, he cannot remember having seen more than two or three cases in the wards, but during the same time his private cases had been nearly one hundred. (Dr. Ransome is speaking of diphtheria, as he says that in croup he has found only a little ulcer in the larynx, no false membrane. The peculiar circumstances possibly account for this exceptional experience).

Dr. SHEARMAN, of Rotherham, as physician to the Rotherham Hospital for six years, has only seen one case of croup, speedily cured, and no diphtheria.

Mr. LAWSON TAIT speaking of cases in which he has done tracheotomy, states that in those where the operation was for false membrane, solely below the epiglottis (believed to be true croup), and where the false membrane was expelled and seen, they recovered with no paralysis, but where false membrane extended over the fauces, well-marked "diphtheritic paralysis" was seen in several. His experience seems to have been pretty large and unbiassed.

Dr. CHAS. BELL, of Edinburgh, has had large school experience, and also private practice. He strongly denies the identity of croup and diphtheria, as regards mode of extension and treatment, and epidemic nature of diphtheria. He states that in croup the false membrane *ascends*, in diphtheria *descends*, and the latter may always be checked by local application (Condy's fluid).

Dr. PYE-SMITH does not say that he has had any experience of membranous laryngitis or acute catarrhal suffocative laryngitis apart from cases of undoubted diphtheria. This he states definitely with regard to adults. He mentions three fatal cases due to diphtheritic contagion in adults under forty. "In all the fatal cases he has seen death appeared to be directly due to extension of inflammation through the bronchial tubes, producing dyspnœa." He believes "that in hospital practice in London diphtheria is more common than croup, even in children." He believes that the characters which suffice to distinguish diphtheritic cases are the appearance and structure of the membrane, its locality, the general symptoms, onset, fever, albuminuria, and paralysis; aided during life by (1) the age of the patient, (2) the occurrence of more than one case at a time. But when membranous laryngitis is once established from whatever cause, and spreads downwards, the subsequent course of the local disease in the air-passages is much the same. He does not regard all membranous laryngitis as diphtheritic.

Dr. W. SQUIRE speaks from considerable experience of parochial institutions for children, and other parochial experience. He states that the cases of croup were all brought into the infirmary from the poorer and more densely crowded parts of the parish. An inspection of the home conditions under which they occurred showed that the rule was that several children slept with their parents in very limited, and often insufficient, air-space, and that there were no instances of more than one child suffering in a family. At the same time there were no cases of croup amongst the children in the workhouse and parochial schools, who were all together in one house. Vaginal diphtheritis was once or twice epidemic. Dr. Squire also states that he had four cases of croup in one month at the St. George's

(Hanover Square) Dispensary (this was at the end of March), and no other cases in the rest of the year.

Dr. SANSOM has had large experience of children at the North-Eastern Hospital for Children, and very strongly distinguishes croup from diphtheritic laryngitis, stating the views usually received by the adherents of this view, and insisting very strongly upon the fact of non-contagion in the cases of laryngeal membranous exudation not of diphtheritic origin. His experience includes some twenty cases of "croup."

Dr. WELLS, of Gualaguaychu, speaks with hospital experience, and gives valuable facts, but more under previous questions.

Dr. GRIMSHAW, of Dublin, has had no experience of croup in hospital practice.

Dr. ALOIS MONTI forty years' hospital experience.

Dr. BAKEWELL, of Trinidad, not *especially* hospital.

Dr. BATHURST WOODMAN speaks from experience of about fifty cases at the London Hospital, North-Eastern Hospital for Children, and general practice in various parts of England. Dr. Woodman believes that all croup is laryngeal diphtheria, but states that he has seen false membrane from scalds, poisons, &c.

The late Dr. LAYCOCK thus sums up his experience: "I have been Clinical Professor for twenty-one years in the University of Edinburgh, and as Professor of the Practice of Medicine I have had to weigh the evidence given on both sides of the question. I have come to the conclusion that the two diseases are wholly different. I have seen cases of diphtheria in which the most careful observation could detect only very slight specks on the fauces to be followed by diphtheritic paralysis. Again, I have known the so-called hospital sore throat, with primary symptoms of no importance, become rapidly fatal as diphtheria, apparently from palsy of the respiratory system. Diphtheria is a highly proteiform disease, and is manifested in every degree of severity according to the state of the individual and his surroundings, and in many cases there is little faucial disturbance. On the other hand, croup is not proteiform, but is—croup."

Dr. OSCAR WYSS says: "My experiences are, *diphtheritis* is a contagious specific disease; *croup* is only a very high degree of inflammation, with exudation of fibrinogenous matter."

Dr. WILKS has given the results of his observations in reply to questions 8, 9, and 10, in the following words:

"I should say, therefore, that the symptoms alone and the post-mortem appearance of the affected parts are not sufficiently characteristic to enable us to distinguish between the two supposed different forms of the membranous disease. My opinion was long undecided about the anatomical differences of the two, but I now think that if there be a difference it is not sufficiently marked to frame a diagnosis. I would not allow, however, on general pathological grounds that similar morbid states establish

an identity of cause, for by way of illustration I might mention that a case of idiopathic dysentery might not be distinguishable from the inflammation produced by a poison. On the post-mortem table we might see two bodies whose death was caused by pneumonia or peritonitis, the one the result of septicæmia and the other of direct injury, the pathology in those two cases being of a totally different kind. It is true that further knowledge might enable us to discern differences in the blood of each, but at the present time, with the simple anatomical facts before us, we must admit identity of appearances having different causes for their production."

"I would also remark that I think we cannot restrict the question of membranous or croupous inflammation to a defined line at the termination of the trachea. It is true that in most cases the membrane occupying the larynx and trachea ends at the bifurcation, and then gradually becomes loose and passes into a mucous secretion, but I have seen cases of undoubted diphtheria and others of questionable origin where the membrane has passed into the bronchial tubes, so that they have been ejected in an arborescent form, or have been found occupying all the bronchial ramifications after death. It is not likely that a distinct line should separate two different morbid processes. I should say, therefore, that the question cannot be confined to the larynx and trachea, but must include the whole of the air passages."

"The pathological question, then, is—*Is there such a thing as an idiopathic membranous inflammation of the air-passages?* This has been unanimously believed up to the present time, but now is denied, the declaration now being that a membranous inflammation of the air-passages is ALWAYS *due to the influence of an external specific agency.*"

"My own view has reached at present thus far—that membranous inflammation of air passages may be due to an external specific cause known as diphtheria, and very often when it is regarded as idiopathic subsequent events will show its specific origin. I could not, therefore, in any given isolated case say, from symptoms or post-mortem appearances of air-passages, that it was not of diphtheritic origin. At the same time, because of this possible fact, I do not think we are yet in a position to justify the statement that it must be so, or that it may not have occurred spontaneously, for the ordinary proof of diphtheria is wanting in a large number of cases. Although I am not wedded to any old opinion, I cannot but hesitate to hurriedly deny a doctrine universally held. It must be known that every pathologist in the world (I believe) has described two forms of idiopathic inflammation of the air-passages, the catarrhal and croupous or membranous, and that every clinical physician has assented to this division. The new doctrine is an absolute denial of this, and that only one form, the catarrhal, exists; the other, the

membranous, is due, it is said, to an extraneous cause. It is not said that the inflammation may be sometimes of one kind or the other, for this is the old and generally received opinion, but that there is no such thing as an idiopathic membranous inflammation of the air passages."

"Modern observation has clearly shown that the latter is frequently the result of an external cause, but are we from this fact to deny the possibility of its spontaneous occurrence? It may certainly occur as a result of artificial irritation, as from steam, but this is objected to as too violent a cause."

"Denying, therefore, that the proof can lie in the consideration of symptoms or anatomical changes, it must be founded on clinical observation over a long series of years. If it be found that a large number of cases of membranous croup occur in isolated spots without the presence of marked diphtheria in the neighbourhood, and in these cases there be no albuminuria or other marked constitutional disturbance, I see no reason why they should not be regarded as of idiopathic origin."

To facilitate reference, and to avoid unnecessary repetition of names, the following list of some of those who have sent replies is given. The number appended to each name is that which was attached to the copy of questions sent to each Fellow, and served as a reference.

- 19. Dr. BAKEWELL, Trinidad.
- 23. Dr. WHYTE BARCLAY, St. George's Hospital.
- 28. Dr. GILLMANN BARRATT, Bayswater.
- 34. Dr. LIONEL BEALE, King's College Hospital.
- 43. E. U. BERRY, Esq., Gower Street.
- 60. Dr. R. L. BOWLES, Folkestone.
- 70. CHAS. BROOKE, Esq., F.R.S., London.
- 79. Sir GEO. BURROWS, Bart., M.D., London.
- 140. Dr. J. E. CUREEY, Lismore.
- 158. Dr. LANGDON DOWN, London Hospital.
- 162. Dr. DYCE DUCKWORTH, St. Bartholomew's Hospital.
- 167. Dr. CLEMENT DUKES, Rugby.
- 175. Dr. EAGER, Bristol.
- 199. Dr. FITZPATRICK, Sussex Gardens.
- 205. Dr. LONG FOX, Clifton.
- 209. J. G. FRENCH, Esq., Maida Hill.
- 216. GEORGE GASKOIN, Esq.
- 217. JOHN GAY, Esq., Great Northern Hospital.
- 226. Dr. W. T. GREENE, Old Kent Road.
- 231. Dr. GRIGG, Westminster Hospital.
- 242. J. F. HARDING, Esq., Uckfield.
- 269. M. BERKELEY HILL, Esq., University College Hosp.
- 284. Dr. E. HUMBY, St. John's Wood.
- 291. Dr. CHAS. HUTTON, Belgrave Square.

- 299. Dr. JACOBOWICS, Vienna.
- 311. Dr. PRICE JONES, Surbiton.
- 315. Dr. KENDELL, Wakefield.
- 318. J. A. KINGDON, Esq., Lothbury.
- 326. Dr. T. LAYCOCK, F.R.S.E. (the late), Edinburgh.
- 327. JESSE LEACH, Esq., Heywood.
- 336. Dr. OSCAR WYSS, Zurich.
- 337. T. LONGMORE, Esq., C.B., Netley.
- 339. Dr. LOWNDS, Egham (late of Bombay).
- 340. J. LUKE, Esq., London Hospital.
- 346. Dr. R. M'DONNELL, St. Steven's Hospital, Dublin.
- 359. JOHN MARSHALL, Esq., F.R.S., University College Hospital.
- 373. Dr. NORMAN MOORE, St. Bartholomew's Hospital.
- 388. Dr. NICHOLLS, Chelmsford.
- 393. Dr. O'CONNOR, March.
- 400. Dr. O'NEILL, Lincoln.
- 404. Sir JAMES PAGET, Bart., F.R.S., London.
- 420. Dr. A. J. POLLOCK, Charing Cross and Foundling Hospitals.
- 421. G. D. POLLOCK, Esq., St. George's Hospital.
- 423. Dr. G. V. POORE, London.
- 435. Dr. RANKE, Munich.
- 436. Dr. RANSOM, Nottingham.
- 466. Dr. W. H. O. SANKEY, University College.
- 467. Dr. A. E. SANSOM, London Hospital.
- 475. Dr. W. SHARP, Rugby.
- 478. Dr. SHEARMAN, Rotherham.
- 492. W. SMITH, Esq., Clifton.
- 496. Dr. REGINALD SOUTHEY, London.
- 517. LAWSON TAIT, Esq., Birmingham.
- 531. Dr. TILT, London.
- 534. Dr. TREND, Southampton.
- 560. Sir THOMAS WATSON, Bart., F.R.S., London.
- 567. Dr. HARRY WELLS, Gualaguaychu.
- 576. Dr. SAMUEL WILKS, Guy's Hospital.
- 580. Dr. C. T. WILLIAMS, Brompton Consumption Hosp.
- 585. Dr. WILLIS, Barnes.
- 590. G. L. WOOD, Esq., Bath.
- 594. W. C. WORTHINGTON, Lowestoft.
- 596. Prof. MACLEAN, Netley.
- 598. Dr. CHARLES BELL, Edinburgh.
- 602. Dr. W. YEATS, Bankfoot, near Perth.
- 610. Dr. PYE-SMITH, Guy's Hospital.
- 614. Dr. GRIMSHAW, Dublin.
- 615. Dr. PAINTER, Beaufort Gardens, London.
- 618. Dr. T. STEVENSON, Guy's Hospital.
- 622. Dr. ALOIS MONTI, Vienna.

- 624. Dr. W. SQUIRE, London.
 - 629. Mr. STRETTON, Kidderminster.
 - 631. Dr. THORNE THORNE, London.
 - 634. Dr. P. DUNCAN, Croydon.
 - 636. Dr. STEPHEN MONCKTON, Maidstone.
 - 638. Sir J. ROSE CORMACK.
 - 639. Dr. GOWAN.
 - 641. Mr. G. B. IRVING, Stanmore.
Dr. MACPHERSON.
 - 645. Mr. J. SOUTHERN, Ludlow.
Dr. BEEBY, Bromley.
Dr. BOWLES, Folkestone.
Dr. HOGGAN, London.
Dr. WHITEHEAD REID, Canterbury.
- And some others, merely formal.

APPENDIX II.

REPORT ON THE HISTORY AND EARLY LITERATURE OF THE SUBJECT.

IN reviewing the historical evidence bearing upon the present question many difficulties present themselves, in consequence of the loose manner in which the term "croup" has been employed, and of the absence of the word altogether in medical or any other scientific writings before the publication of Dr. Home's treatise. The word "diphtheria" or diphtheritis never appears until after the publication of Bretonneau's Memoir in 1826. Nevertheless, the disease to which the word is attached has probably existed from all antiquity, and its history must be traced under other names. Another difficulty to be most carefully avoided is the following. The present investigation being directed to prove the identity or non-identity of "membranous croup" and "laryngo-tracheal diphtheria," and not to establish the diagnosis between a disease essentially characterised by the presence of false membrane, and some other disease in which there is no false membrane at all, it is necessary to exclude carefully from consideration many of those cases which pass popularly under the designation of croup, such as laryngismus stridulus, non-membranous laryngitis, and, it may be added, cases of tumours in the larynx, foreign bodies in the larynx, aneurisms pressing upon the recurrent laryngeal nerve, &c., in all which there may be what are called "croupy" symptoms. The inquiry, therefore, for present historical purposes, must be strictly confined to those cases where there is distinct evidence of a false membrane in the larynx and trachea, in either or in both.

Bretonneau, in all his writings, draws a distinct line of demarcation between "diphtheria" and ordinary laryngeal inflammation, on the one hand, and laryngismus stridulus on the other; and the only question to be determined is really whether the disease he described as tracheal, or laryngo-tracheal, diphtheria is different from what, before his writings appeared, was known as "membranous croup." He himself declares that they are exactly the same, and he also traces the history of what *he* calls "tracheal diphtheria," and what *others* call "membranous croup," to a very remote antiquity, long before either of these names was employed. The following historical summary will throw some light upon this difficult question; and it must be mentioned at

the outset that every possible care has been taken to eliminate from the field of investigation all cases in which there was no false membrane.

That a disease characterised by the presence of a false membrane in the larynx or trachea, or in both, and attended by great fatality, has prevailed in the world from the remotest period is in the highest degree probable, but the Greek writings on medicine, which are amongst the earliest medical treatises in existence, are too vague in their descriptions to enable us to affirm that any disease exactly resembling that now known as membranous laryngitis was known to or observed by the Greek physicians. The passage in Hippocrates, which is sometimes referred to as relating to diphtheria, contains only some vague descriptions of a complaint in the throat which would apply as well to cancer, or tonsillitis, or malignant scarlatina, or tertiary syphilis, as to diphtheria or membranous croup. The description given by Aretæus, although more definite than that of Hippocrates, is far from conveying to the mind any distinct idea of diphtheria, and a careful examination of his chapter in the original Greek, *περὶ τῶν κατὰ τὰ παρίσθμια ἑλκῶν*, leaves the reader quite in doubt whether the author described an ordinary "ulcer" or a membranous formation in the fauces, but the critical study of the original Greek words rather leads to the former view. The writings of Celsus contain no more definite descriptions of diphtheria than those of Hippocrates, and they are even less definite than those of Aretæus.

It would appear that an epidemic of tracheal diphtheria prevailed in Paris towards the end of the sixteenth century, for Baillou (Ballonius), writing in 1576, writes thus: "*Chirurgus affirmavit se secuisse cadaver pueri ista difficili respiratione et morbo (ut dixi) incognito sublato; inventa est pituita lauta, contumax, quæ instar membranæ cujusdam arteriæ aspera erat obtenta, ut non esset liber exitus et introitus spiritui externo: sic suffocatio repentina.*"

But at the commencement of the seventeenth century there is no doubt whatever that a disease exactly resembling the tracheal diphtheria of Bretonneau prevailed extensively in Spain, and was accurately described by the Spanish physicians of that period. Their names are Herrera (Christopher Percy de), '*De Essentiâ &c., Morbi Suffocantis Garrotillo Hispanæ appellati*,' published at Madrid in 1615; Fontecha (J. A. de) '*Disputationes Medicæ*,' published at Alcalá in 1611; and Villa Real (J. de), '*De Signis &c., Morbi Suffocantis*,' published also at Alcalá in 1611. The following description, out of many given by these writers, is quite conclusive as to the nature of the disease they witnessed. Villa Real describes "*quasdam velut membranas cingentes fauces, et tali constantes modo substantiæ, ut si propriis manibus tendas, videas ejus partes cedere, quas,*

si desinas, videas refluere, propriumque adquirere locum, non secus ac si corium madidum aut membranam madidam tendas et sinas. Hoc experientiâ didici," he says, "tum in viventibus excretâ causâ per os, tum in morientibus factâ anatomiâ." Here he distinctly indicates the false membrane resembling moist leather, stretching and relaxing under the hands, and he states that he has seen it both when thrown up by the mouth and when examined in the bodies after death.

About the same period epidemics of exactly the same nature occurred in some parts of Italy, and they are described by Carnevale, in a treatise '*De Epidemico Strangulatorio Affectu*,' published at Naples in 1620, and by Nola, in a treatise '*De Epidemio Phlegmone Anginoso grassante Neapoli*,' and published at Venice in 1620. A similar epidemic is described by Cortesius as raging at Messina, in Sicily, in 1625.

There are not many more distinct records of the disease in the seventeenth century, but in the eighteenth they are abundant. An epidemic is described as prevailing at Cremona, in a small work called '*Lettere Mediche*,' published by Martius Ghisi in 1749; another was witnessed in Sweden, and was described by Wilcke in a work entitled '*De Angina Infantum*' published at Upsala in 1764; a most graphic account of one occurring in Picardy, in France, was published by Marteau de Grandvilliers in a work entitled '*Description des Maux de Gorge Epidémiques et Gangréneux qui ont régné à Aumale et dans le voisinage*,' Paris, 1768; and similar epidemics were described by Bard in 1771, as having occurred at New York, and by Starr, as having broken out in Cornwall.

In all the epidemics just referred to it must be distinctly understood that in every instance the false membrane in the windpipe is clearly indicated by the respective authors. As a specimen of the description, the following may be quoted from Wilcke's Dissertation on the '*Disease in Sweden*.' When describing the appearances found in a post-mortem examination made by Dr. Rolandus Martin, he writes: "*Asperam arteriam intus undique singulari inductam membrana observavit quam sponte fere nexu omni solutam, peculiaris tubi instar, extraxit; crassiore; grisea et ex putredine laciniosa qua cavum sui spectabat; qua vero asperæ arteriæ adhæserat, sanguineo-purpurea. Quo longius in pulmones descenderet, eo pallidioris fuit ruboris, et in subtilissimis quidem bronchiorum ramis prorsus albicans, speciem præbuit membranæ, quæ ovi putamen intus investit.*"

The epidemics described by Fothergill and Huxham in the last century, and which have been sometimes regarded as proving the prevalence of diphtheria, are here purposely omitted from consideration, because it is quite evident, from a perusal of the works of those authors, that the diseases they described were some

aberrant forms of scarlatina, with which tracheal diphtheria has no necessary connection.

In 1765 Francis Home described a disease which he witnessed on the east coast of Scotland. He appears to have known nothing of the works of any of the authors already cited, and he considered the disease he witnessed to be a new one, and he described it under the name of "croup," a Scotch word first introduced into medical literature by Dr. Blair, of Cupar Angus, in 1713. Home's cases are only twelve in number, and of these three appear to be cases of non-membranous laryngitis. Nine of them, however, are undoubtedly instances of membranous laryngitis, or at least Bretonneau considered that they correspond exactly to the description which he afterwards gave of laryngo-tracheal diphtheria.

At the very commencement of the present century, namely in 1801, appeared the essay 'On Cynanche Trachealis or Croup,' by Dr. Cheyne, and this work requires very careful consideration in connection with the present Report, because it is from Dr. Home and Dr. Cheyne that the word "Croup" has been introduced into the language of medicine, and the descriptions of these two authors have been adopted by most subsequent writers. His cases are only ten, and, out of these, three are not his own. Five of them terminated favorably, and as Dr. Cheyne believes, in consequence of bleeding and purging, but, as in them no false membrane is described, it may be fairly a question whether these cases were not instances of non-membranous laryngitis. Five cases were fatal in spite of all treatment, and in four of them the false membrane is not only described, but is very admirably drawn and coloured, the artist being no less a master in pictorial art than the afterwards distinguished Sir Charles Bell. The examination of these plates leaves no doubt whatever that membranous exudation existed. Cheyne appears firmly to believe that the false membrane is the result of the inflammatory action, and that its appearance may be prevented by bleeding and purging.

After the appearance of Dr. Home's and Dr. Cheyne's treatises the word "Croup" began to be employed to designate the disease characterised anatomically by the presence of a false membrane in the larynx and trachea, and which had been previously known under the names of Garrottillo, Morbus Strangulatorius, Male in Canna &c. The occurrence of the death of the grand-daughter of the French Empress Josephine, in 1807, from this affection,¹ induced the Emperor to offer a large prize for the best essay on the disease, then known as "Croup;" and the chief object of this prize was to lead to some suggestions as to the best mode of cure.

This era is important in the present investigation, because the occasion produced a multitude of essays, not only from French,

¹ A tubular false membrane was found in the child's body after death.

but other authors, who were attracted by the great value of the prize and by the hope of distinction, and the award was finally divided between two aspirants, namely, Albers, of Bremen, and Jurine, of Geneva. The essay of Albers is written in Latin, and is entitled '*Commentatio de Tracheitide Infantum, vulgo Croup vocata*,' and it was published at Leipzig in 1816. The essay of Jurine was written in French, but his work is very scarce, and is only met with in a German translation. Other essays on the same subject sent in for competition were by Double ('*Traité du Croup*,' Paris, 1811), by Vieusseux, ('*Mémoire sur le Croup*,' Genève, 1812), Valentin, ('*Recherches historiques et pratiques sur le Croup*,' Paris, 1812), and some other treatises were written (although not for the competition) on the same subject by Desruelles, Guibourt, Caillou and others.

Although the essays thus written all adopted the name "croup" as indicating the disease they described, it is very evident from a perusal of the works that the authors invariably described at least three different diseases under the same word, and that all throat affections, in fact, were by them called "croup," especially if they occurred in children. Albers, who gained the prize, and who may be supposed to write authoritatively on the subject, clearly regards croup as a disease offering many and very different characters, according (1) as it is or is not accompanied by a false membrane, or (2) as the nervous or inflammatory symptoms preponderate; and although he expresses his opinion that "*morbis noster consistit in membranæ pituitosæ tum laryngis, tum arteriæ asperæ et ramorum ejusdem inflammatione*," yet he is evidently of opinion that the "lymphæ plastica" which is developed by that inflammation may be arrested in its development, or absorbed after its effusion, by appropriate medical measures. His own success in the treatment was extraordinary, and in the few cases where the patients died the result, he says, was entirely due to the fact that the remedial measures (chiefly bleeding and mercury) were not adopted sufficiently early or with adequate vigour. The history of one of his unsuccessful cases "clearly shows us," he says, "*nos apertè docet, remedia statim ab ejus principio maxime festinanda esse, propterea quod periculum in levissima versetur mora*." It is unnecessary in the present day to comment upon these views, and it is quite evident that the majority of cases said to be so successfully treated by Albers were cases of non-membranous laryngitis, and not of the disease which forms the subject of the present Report.

The essay of Jurine, which, as before mentioned, is very scarce, and only exists in England as a German translation by Albers, advocates exactly the same views as the latter author, and places the same reliance on antiphlogistic treatment. Jurine gives from his own experience twenty-eight cases of "Croup," of which

only three were fatal—a result which almost in itself proves that the author did not witness cases of membranous laryngitis, the mortality of which is notoriously excessive. Even of the fatal cases, it is more than probable that two of them, judging by the description, were instances of non-membranous laryngitis. Jurine evidently believes that the antiphlogistic treatment, as it is called, prevents the formation of the false membrane, “Die erste Indication bei dieser Krankheit ist nicht das Koncrement wegzuschaffen, sondern dessen Bildung zu verhüten.” He therefore strongly recommends bleeding to effect this object. Like all the other essayists, Jurine makes no attempt whatever to show that there are two diseases, both of which produce a pseudo-membranous exudation in the larynx and trachea.

But the other essays sent in for competition are almost exactly of the same character as those of Albers and Jurine, except, perhaps, that they do not all so loudly and confidently extol the value of curative measures. They all, in fact, regard “croup” as divisible into three categories, namely, 1, where a false membrane is found in the windpipe; 2, where there is no false membrane, but where inflammatory symptoms exist; and, 3, where there is a preponderance of “nervous” symptoms. In other words, they include under “croup,” 1, membranous laryngitis; 2, non-membranous laryngitis; and 3, laryngismus stridulus.

Excluding non-membranous laryngitis and laryngismus stridulus, and endeavouring to extract from the French essayists of the period now referred to, any information as to the divisibility of membranous croup into that caused by common inflammation and that due to epidemic influence or contagion, the inquiry is hopeless and the results are negative. The authors all regard the false membrane as the result of an inflammatory process, and their curative measures are directed with a view to subdue the inflammation, and thus either to prevent the formation of the false membrane, or to cause its absorption when it has been developed. Whether the means they recommended, which consisted in copious bleeding, the administration of mercury, and other so-called antiphlogistic measures, ever really effected these objects it is scarcely worth while in the present day to inquire.

While the scientific features of “croup” were thus represented by a large number of French writers, the epidemics of Tours presented themselves to the notice of Bretonneau, and it is necessary, in an historical point of view, to fix accurately the dates of the French writings just referred to, and that of the outbreak of the first Tours epidemic. It will be seen that the ‘Essays’ appeared in 1811, 1812, and 1816, and the first epidemic witnessed by Bretonneau occurred in 1818. Bretonneau, therefore, who was well acquainted with medical literature, must have been fully aware of the existing doctrines on the subject of

"croup," and he, indeed, adopted the word himself. But he saw that the "croup of his contemporaries comprised at least three different diseases, as has been indicated in a previous page, and he therefore proposed the word "diphthérite" to indicate that form of croup which was attended by the presence of a false membrane in the larynx and trachea. He himself first regarded the disease as inflammatory, and hence the name "diphthérite" (which, however, he subsequently changed for "diphthérie"), and he accordingly, at first, and under the influence of the then prevailing doctrines, endeavoured to combat the symptoms by bleeding, mercury, and other so-called antiphlogistic measures. But he soon abandoned them all as not only useless but injurious, and regarded the false membrane, not so much in the light of an inflammatory exudation, as a specific development of the disease arising from infection, and thus resembling in its nature the pustules of smallpox, the rash of typhoid, &c.

It is quite hopeless to search the writings of Bretonneau for any distinction between "pseudo-membranous croup" and "laryngo-tracheal diphtheria," for he evidently regards them as identical, and expresses himself repeatedly to that effect. Besides separating (1) "tracheal diphtheria" or "croup," or "vrai croup," as it is called by some very modern French writers, from (2) ordinary laryngitis and (3) laryngismus stridulus, *Bretonneau established the fact that in the majority of his cases of membranous laryngitis the affection of the trachea was attended by the appearance of the characteristic leather-like membrane on the fauces*, the latter appearance thus giving warning of the dangerous and generally fatal issue. He admitted, however, that in a certain number of cases the tracheal affection was not attended by any exudation on the fauces, and this form of disease is fully recognised by modern French writers, who call this form of diphtheria the "croup d'émblée."

In his earlier memoirs on diphtheria (1821-26) Bretonneau made it his principal object to prove that that disease and croup are identical. But it is worthy of note that both Home and Cheyne were perfectly acquainted with the fact that the disease which they described was liable to be confounded with one which affected the larynx secondarily, having its original seat in the fauces. One of these writers, quoting Dr. Starr's account of the "*morbis strangulatorius*" in Cornwall (which was epidemic diphtheria in its most typical form), says that that complaint "appears more nearly allied to the malignant sore throat, although it sometimes attacked the trachea." And the other commences his chapter on diagnosis by remarking that he had seen several children whom he would have supposed to be suffering under the second stage of croup had he not discovered sloughs upon the tonsils and uvula. Bretonneau, however, showed that in some cases of diphtheria the affection was limited to the air

passages below the epiglottis. One case of this kind is recorded in his fourth memoir (Case 45). It is that of a child, aged one year, which was under the charge of a nurse living near Tours. Bretonneau himself points out that in this town there had not for many months been a single subject attacked with epidemic angina, with the exception of the patients admitted to the hospital. The disease was, however, prevailing at the hamlet of Chenusson eight French leagues to the north of Tours, and on inquiry the fact was elicited that the nurse was a native of Chenusson, and aunt to a boy who had died of diphtheria five or six days before the child was attacked. She declared that the fear of the contagion had hindered her from receiving any one who had been in communication with patients suffering from the disease; but (it is added) "in the very terms of her denial proof was found that she had been in communication with several of them." It is worthy of attention that the proof of the diphtheritic nature of this case was thus incomplete, although there can be little doubt about the matter. Bretonneau goes on to say that this was the second time, and in the proportion of one to thirty, that he had met after death with diphtheritic inflammation limited to the air-tubes. It is therefore evident that whatever may be the nature of membranous croup, it was at that time in France a rare disease.

Bretonneau's doctrine as to the diphtheritic nature of croup was in due course adopted by his pupil Trousseau, by Guersant, Barthez, and almost all the other leading French physicians. In England it was for a long time repudiated by every medical writer, but within the last few years it has met with a much more favorable reception. The late Dr. Hillier advocated it in 1862, and since then Dr. Johnson, Dr. Semple, and Sir J. A. Cormack have maintained it, and Sir W. Jenner has withdrawn his previously expressed opinion that the two complaints are distinct.

In adopting the view that membranous croup is always a form of diphtheria, Bretonneau was, of course, obliged to draw a sharp line of distinction between that affection and all milder forms of laryngeal inflammation unattended with the formation of false membrane, and the same opinion has necessarily been maintained by all those who have since adopted the doctrine. Bretonneau's name for the non-membranous affection was "stridulous angina," it has also been called "stridulous laryngitis," "inflammatory croup," and "infantile laryngitis."

Now, it is important to note that the English writers of the last century were well acquainted with the fact that croup was liable to be mistaken for another affection which they designated "spasmodic" or "spurious" croup. We have not been able to discover who originally pointed out the peculiar characters of this disease, but they are fully set forth in a paper which Mr.

Field, Apothecary and Secretary to the Medical Society of London, read before that body in 1796. The most distinctive features are the suddenness of onset of the disease, the alarming nature of the symptoms from the very commencement, and its tendency to return again and again in the same patient.

Now Cheyne, in the second edition of his work, discusses at considerable length the relation between the disease of Home, and this "spurious" or "spasmodic" affection, with which he was acquainted through the writings of Field, and of Ferrier, a physician of Manchester, who had published an essay on the subject in 1810, and he comes to the conclusion that there are no just grounds for admitting two kinds of croup. The affection in question occurs, he says, "in those families which are subject to genuine croup ; it arises from the same exciting cause (exposure to cold) ; it prevails during the warm weather."

On referring to the cases of which Dr. Cheyne records the details, it appears that all those among them which terminated in recovery presented more or less clearly the characters of the "spurious" affection. The comparative harmlessness of this form of croup has since been pointed out by other writers.

APPENDIX III.

HISTORICAL SKETCH OF ANATOMICAL DISTINCTIONS WHICH HAVE BEEN DRAWN BETWEEN CROUP AND DIPHTHERIA.

THE history of the terms Croup and Diphtheria, and of the conceptions connected with them, has been so different in Germany and England, that they will most conveniently be treated of quite separately.

HISTORY OF THE ANATOMY OF DIPHTHERIA IN ENGLAND.

Whenever an anatomical distinction between croup and diphtheria has been drawn in this country, it has always started with the assumption that the croupous membrane is a simple fibrinous exudation, and with this the diphtheritic membrane has been compared, and it has been sought to establish a difference in two respects—(1) in the presence of some parasitic vegetable growth; (2) in the diphtheritic membrane being composed of cells without fibrine.

1. *Vegetable growth.*—Laycock, in a lecture at Edinburgh, May 29th, 1858, regarded diphtheria as the product of a fungus, viz. the *oidium albicans*. Hillier, on the other hand, pointed out that while the false membrane consisted chiefly of cells, *i.e.* mucous corpuscles and epithelial elements, with few fibres, a fungus might be found in some cases, but this was *Leptothrix buccalis*, not *oidium*, and of no pathological importance.

Other observers published cases in which no fungus elements were found, and the theory of parasitic infection was generally dropped in this country until recent years.

2. *Cellular structure.*—When cases of diphtheria began to be observed in this country during the great epidemics of 1855–59, &c., it seems to have been at first assumed that the minute cha-

acters of the diphtheritic false membrane were the same as those of the croupous, which had been always regarded as composed chiefly of fibrine. Some observations were however published, such as those of Dr. George Harley ('*Path. Trans.*,' xii, 241), to show that the diphtheritic membrane consisted of cells and not of fibrine.

An apparently similar but really different view has been held by Wagner in Germany, and confirmed by MM. Cornil et Ranvier ('*Hist. Path.*,' p. 89).

More recently Dr. Murchison published a case of diphtheria extending to the trachea and bronchi, in which he reiterated the account of the diphtheritic false membrane as composed of cells "everywhere made up of modified epithelial cells, and containing no fibrillated tissue." ('*Path. Trans.*,' xxii, p. 36)

Hillier ('*Diseases of Children*,' 1868) describes the false membrane of diphtheria as, when examined microscopically, being found to consist of altered epithelial cells, of granular corpuscles and nuclei. In the deeper layers pus globules and blood-disks are often seen. Fibrillation, such as occurs in fibrinous exudation, is sometimes seen on the under layers of the deposit.

Jenner ('*Diphtheria*,' 1861) describes the product upon both the pharynx and the larynx as "lymph," and speaks of it as varying much in consistence in different cases, sometimes as soft as cream, sometimes as resembling wash leather. The softer varieties consisting of free granular corpuscles and epithelium, the tougher of fibres such as are seen in the buffy coat of blood coagula.

He speaks of the lymph as often difficult to separate from the pharyngeal mucous membrane; but in the larynx and trachea a distinct separable membraniform layer. The exudation may begin in the larynx, and spread downwards to the trachea and bronchi, without involving the pharynx.

In the pharynx, the inflammation is not limited to the mucous membrane, or even to it and the submucous tissue, for the deeper parts are thickened and toughened.

Vegetable growths have no doubt been occasionally seen in diphtheritic exudation, but being often absent, play no essential or important part in the cases of diphtheria seen by Jenner.

No anatomical distinction is given between croup and diphtheria, but warning is given against confounding diphtheritic exudation with the specific disease, diphtheria.

According to Greenhow ('*On Diphtheria*,' 1860) the exudation varies in consistency from a pultaceous or almost liquid exudation, to a firm, consistent and more or less elastic membrane. The elastic form of false membrane is not unlike the exudation poured out from an inflamed serous membrane. Sometimes the exudation is not membranous, but dry and granular. Examined under the microscope, it is found to consist of coagulated fibrine and epithe-

lium; the latter usually more abundant in the outer portion of membrane, while the deeper portion is more purely fibrinous. Exudation cells are often intermixed with the fibrillar tissue. Low forms of cryptogamic plants occasionally found, but not invariably, and also found on unhealthy mucous surfaces in other diseases. Greenhow, however, describes necrosis and sloughing as frequent pathological appearances of diphtheria, with or without the presence of false membranes.

Further observations on the anatomy of the false membrane in diphtheria, were made by Bristowe (1859), Sanderson (1859), Harley and others. The two former found fibrine to be an element in the false membranes. The existence of this constituent was denied by Harley and others. No comparative observations appear to have been made in cases of undoubted croup.

Squire, in 'Reynolds' System' (vol. i, p. 259 and p. 397), in 1866, speaks of the croupous membrane as different from that of diphtheria "both in its chemical and physiological relations. It is not simply fibrine, but consists of effused lymph, in which the presence of albumen can always be chemically demonstrated. Microscopically it is a mass of cystoid corpuscles." Also it is not the result of interstitial change in the mucous membrane, but is an exudation. Other observers appear not to have traced the chemical distinction, or have thought it unimportant; and most would repudiate the notion that the diphtheritic membrane is exclusively or specially fibrinous.

None of these observers seem to have drawn any general distinction between laryngeal or tracheal products on the one hand, and pharyngeal or faucial on the other; but unless this distinction be kept in mind, the distinction of croupous and diphtheritic products becomes ambiguous or impossible.

It is to be noted, in comparing observations quoted above with later observations, that at that time, the modern method of making vertical sections through the mucous membrane and the false membrane together does not appear to have been practised.

It is further to be observed that few, if any, comparative observations can be found to have been made on products of croup and diphtheria at the same time, and with the same methods of investigation. In every case the reference has been to an assumed standard or traditional definition of croup. Thus, while several peculiarities have been pointed out in which diphtheritic products differ from croupous products as thus understood, it is probable or almost certain that the same peculiarities would be found in morbid products which have always been regarded as indisputably croupous if examined by modern methods of research.

Various observations on the presence of fungi or bacteria in diphtheria have been of late years published, but need not be

considered here, as they are avowedly either confirmations or refutations of the views of German observers; and no direct comparative investigation has been made of the products of croup.

The testimony of English observers does not seem to supply any anatomical basis for the separation of diphtheritic from croupous products. English pathologists do not appear to have adopted the solution which has been accepted by the Germans, of making croup an anatomical term, applicable even (among other things) to certain products of diphtheria.

HISTORY OF THE ANATOMY OF DIPHTHERIA IN GERMANY.

The history of this subject in Germany naturally divides itself into three periods:

I. The history of the views prevalent before the advent of the cellular pathology, or at the time of its introduction.

II. These views as modified by the researches of Buhl and Wagner on the activities and changes of cells in croup and diphtheria.

III. Recent researches relating to the presence of vegetable growths in diphtheria.

I. The views prevalent in Germany on the anatomy of croup and diphtheria twenty years ago were expressed in the text-books of Rokitsansky and Förster, but had already been influenced by Virchow.

CROUP was regarded as a "fibrinous exudation" effused in a liquid form and coagulating on the surface of the mucous membrane, this being unaltered or nearly so. The false membranes might contain fungus-growth, but this only the ordinary *leptothrix*.

DIPHTHERIA, on the other hand, was described by Rokitsansky as essentially a necrotic process, consisting in infiltration of the mucous membrane, accompanied by exudation, and followed by sloughing; a membranous necrotic mass being formed which may be confounded with a croupous membrane. Rokitsansky regarded this process as identical with that giving rise to "Aphthæ" in the mouth, and on various parts of the intestinal canal. Vegetable parasites are sometimes present, but have no importance. He does not clearly describe any such necrotic affection of the trachea, except as a part of some general disease, as typhus or tuberculosis. It would appear that most, if not all, the false membranes occurring in the air-passages must come under Rokitsansky's definition of croup.

Virchow's views on this subject appear to have been several

times modified, but it seems to have been in great part owing to his influence that the names croup and diphtheria received so wide an extension of meaning as they have had, and now have, in German medical literature. The term croup was applied to all inflammations accompanied by fibrinous exudation on a surface, hence lobar pneumonia became croupous pneumonia; and kidney disease with hyaline ("fibrinous") cylinders in the tubes was called croupous nephritis (though this last explanation was afterwards repudiated by Virchow himself). Again, any necrotic or sloughing process was called diphtheritic, and ordinary dysentery became intestinal diphtheria. Thus, to a German medical reader the names croup and diphtheria convey the ideas, not of laryngeal or pharyngeal disease, but of processes. A correct appreciation of this signification is necessary to understanding the German views on the subject.

Virchow, in 1865, also laid weight on the element of necrosis or sloughing, as essential to diphtheria distinguished from croup, but somewhat extended the views given above.

II. Wagner in his paper published in 'Archiv der Heilkunde,' 1866, p. 481, represents the prevalent views of most pathological anatomists and nearly all clinical physicians as consisting in regarding the pharyngeal diphtheritis and croup of the air-passages as perfect examples of diphtheritic and croupous exudation respectively, being generally regarded as varieties of the so-called fibrinous exudation, differing only in this respect, that the croupous exudation lies on the free surface of the mucous membrane and leaves its tissues unaffected, while the diphtheritic is situated partly on the surface, partly, and more especially, within the parenchyma of the mucous membrane, and causes necrosis of it. The views here stated had, he remarks, been in recent years modified by Virchow, Buhl, and Weber, though these authors were not in agreement with one another.

At starting Wagner takes as the provisional definition of diphtheritis, an affection of the mucous membranes, in which the surface is covered with the well-known grey false-membrane, for the most part adherent to the surface, and under which the mucous membrane itself is more or less infiltrated and thickened. Croup, on the other hand, is provisionally defined as meaning those affections in which the well-known membrane is present but lies loosely upon the mucous surface, and under which the mucous membrane itself is either normal to the naked eye or, at most, hyperæmic.

Wagner further remarks that in the very numerous fatal cases examined by him of simultaneous pharyngeal diphtheria and croup of the air-passages, he found in the back of the mouth as well as in the pharynx the unmistakable characters of diphtheria; in the lower part of the larynx, the trachea, and often also in the bronchi, those of croup; while the upper part of the

larynx sometimes presented an intermediate character, sometimes one more distinctly diphtheritic.

Wagner describes separately:

I. Diphtheria of soft palate and pharynx.

II. Croup of lower larynx and trachea.

I. In the diphtheritic deposit or false membrane, Wagner describes a delicate network of which the fibres were sometimes so delicate as to resemble those of coagulated fibrine, but much more commonly composed of bands of very unequal thickness, generally much broader than threads of coagulated fibrine. Both the thinner and thicker reticulations may be found in the same place, but generally pharyngeal diphtheria showed the thicker; in laryngeal and tracheal diphtheria the finer network preponderates. Transitional forms between the two were found. This network, whether of the finer or the coarser variety, Wagner believed to be never composed of coagulated fibrine, but to be produced by a peculiar metamorphosis of the epithelial cells, which undergo degeneration, and then become partially absorbed so as to produce the appearance of a fibrinous network. The process is compared to that previously described by Buhl (in 1863) as giving rise to the production of croupous membranes. Beside the network-cells of lymphatic type, leucocytes were very generally though not universally present, and were very irregularly distributed. The further details need not concern us here, except as compared with the description given by the same writer of the croupous membrane. In diphtheria properly so called there is, moreover, according to Wagner, constantly an infiltration of the mucous membrane with newly formed cells and nuclei, sometimes amounting to purulent infiltration, and this extends into the submucous tissue; sometimes deeper still into intermuscular connective tissue.

II. The croupous membrane, according to Wagner, as seen in the lower part of larynx and trachea, consists of a close network of delicate threads, enclosing a large number of elements resembling pus corpuscles, the latter being very much more numerous than in the diphtheritic membrane. The origin of this network is, according to Wagner, like that found in diphtheria, from metamorphosis of cells. The mucous membrane itself shows less change, being merely hyperæmic and infiltrated with cells, but chiefly in the upper layers; the lower strata and the submucous tissue being free. The same description applies to the false membranes met with in the bronchial tubes, the only difference being that in the latter cellular structure predominates, and the membranous affection may gradually pass into a simple purulent or mucous bronchitis. Even the small casts "so called fibrinous" found in the smallest bronchial tubes in cases of ordinary lobar pneumonia exhibit the usual structure of croupous membrane.

The above explanation was still further extended by Wagner to so called croupous pneumonia itself. The fibrinous network which fills the alveoli in this disease was explained as derived, almost without a doubt, from the metamorphosis of cells. It was said to show the same variations as the pseudo-fibrinous network of croup or diphtheria, and the pus-corpuscles form in pneumonia as in the disease just mentioned, a considerable proportion of the infiltrated mass.

Finally Wagner describes numerous transitional forms between croupous and diphtheritic membranes, and lays stress upon the fact that both appearances are frequently met with in the same case, at different parts of the respiratory or faucial mucous membrane. A case is called one of pure *croup* where the false membrane lies upon the surface, with little affection of the mucous membrane itself, and, at all events, no purulent infiltration of it, and where the pseudo fibrinous network is composed of finethreads. Pure croup may occur on the tonsils and fauces only, as well as in the air passages, or on both, simultaneously or successively.

The diphtheritic condition was rare in the air-passages and only in a single case found there alone. Other pathologists who in general adopt Wagner's explanation do not recognise the possibility of croupous disease of the pharynx and tonsils, and regard a diphtheritic condition of the air-passages as of the rarest occurrence.

From the above it is clear that Wagner makes no pretension to draw an anatomical distinction between the disease croup and the disease diphtheria. On the contrary, he expressly repudiates any such distinction. The distinction which he draws is between two anatomical conditions merely, without referring to their clinical accompaniments; and even this, as he clearly points out, is a difference of degree. It seems to have been often supposed that if the diphtheritic membrane could be shown to be non-fibrinous, but derived from cells, altered or unaltered, this would of itself distinguish it from the croupous membrane, which has been assumed to be fibrinous. But this opinion, whether correct or not, finds no countenance from the views of those who claim to have shewn the non-fibrinous character of the network found in diphtheritic membranes, since their explanation applies equally to the similar appearance in the croupous membrane.

Buhl, in 1863, promulgated a theory of the nature of croup which should be mentioned, as it has had considerable influence in Germany. He rejected the ordinary *primâ facie* view that fibrine is exuded from the vessels, but spoke of it as formed by a process of secretion within the epithelial cells, which, at the same time produced pus-corpuscles by a process of endogenous proliferation. The same explanation was extended to the production of fibrine within the air-cells in pneumonia, and even to the inflammations of serous surfaces, in which Buhl refused to see

simple exudation of coagulable material, but referred the production of inflammatory lymph to the action of cells. These views do not appear to meet with much acceptance at the present day, especially as regards the identification of croupous membranes with ordinary inflammatory lymph of serous inflammations, but they have had considerable influence on the development of the theory of croup and diphtheria. Buhl, ('*Zeitschrift für Biologie*,' 1887, Band III, S. 367) has attempted to draw a clear histological distinction between croup and diphtheria. He rejects, in the first place, the possibility of a croup of the pharynx, and holds that the question can only arise in case of disease of the air-passages. In croup there is an actual deposit on the surface of the epithelium, consisting of pus cells and a fibrinous cement, which is separated from the mucous membrane by the unbroken epithelium. In diphtheria there is no deposit or new production properly so called. What appears to be such is only a layer of altered epithelium, or of necrotic mucous membrane. The essential difference consists in the condition of the mucous membrane which is, in diphtheria, infiltrated with a new cellular or nuclear growth. There may be, in croup, some infiltration with ordinary pus cells, and they may even be numerous; but there is not that nuclear proliferation seen in diphtheria; moreover, the cellular infiltration is not the important thing in croup, and may be quite absent; while in diphtheria it is never wanting. It is this rapid and abundant infiltration, which, by compressing the blood-vessels, causes local anæmia and necrosis which is characteristic of diphtheria.

Buhl admits that croup and diphtheria may be, and frequently are, simultaneously present, the former in the air-passages, the latter in the pharynx, though more often the affection of the air-passages in such cases is diphtheritic and not croupous. But there is no reason why the diphtheritic infection, like other such infections, should not produce even pure croup of the air-passages.

These views of Buhl's clearly do not distinguish croup and diphtheria as *diseases* by any anatomical (or histological) criterion. With the other points of distinction which he insists upon, we are not now concerned.

It is also noticeable that he appears, in his paper, to have abandoned his former elaborate explanation of the origin of croupous material in the air-passages, lungs, and elsewhere, hence he no longer speaks of the croupous membrane as formed out of cells.

The above extracts represent the state of opinion in Germany, on the histology of croup and diphtheria before the rise of the modern fungous theory of diphtheria, which has thrown other alleged points of difference into the shade.

III. *Third period*.—The literature relating to the alleged existence of vegetable parasites in diphtheria is very voluminous,

and can be here considered solely in relation to the question of the anatomical distinctions of the disease from croup.

Buhl, in 1867, was one of the first who drew attention to the presence of parasitic elements in the diphtheritic slough or false membrane, so small that they had been mistaken for granular fibrine. He leaves it undecided whether the parasite was peculiar to diphtheria, or merely the ordinary *Leptothrix buccilis*; and finally, whether if a peculiar form, it is of any importance in the causation of the disease.

The question was afterwards investigated by very numerous observers, of whom Nassiloff ('Virch. Arch.,' L) and Oertel ('Bayr. Intelligenz Blatt,' 1868, and 'Jahresbericht der Med. Wissenschaften,' 1868, II, 116) described micrococci as occurring in the diphtheritic masses, and traced them into other organs, as well as in the blood. The disease has also been inoculated into animals.

It is to be observed that in Germany, as in England, these observations first related to the ordinary forms of fungi such as are very commonly or normally found in the mouth, and it was soon recognised, that such growths if present, could be of little importance. Afterwards, the more minute forms of *micrococci* or spheroidal bacteria were recognised, which were thought to be of great importance, and with respect to which the only doubt would be whether they were really distinct from the bacteria found in putrifying animal substances generally.

Letzerich differs from other observers in recognising a true fungus with mycelium and spores of the *Hyphomycetes* family as the cause of diphtheria. His views have not been generally accepted, and are important only in the present connection as showing how the assumption of a fungus growth bears upon the explanation of croup.

Letzerich ('Virch. Arch.,' vol. liii, 1871, p. 498, "Beiträge zur Physiologie der Flimmerzellen;" "Ueber Exsudat und Eiterbildung," and 'Die Diphtherie,' Berlin, 1872) holds that in diphtheritis the cause of the affection of the mucous membrane is a fungus which penetrates from without, either destroying the epithelial cells or hollowing them out in large scales, and by penetrating inwards into the tissue of the mucous membrane produces the diphtheritic exudation. Thus it happens that the exudation differs macroscopically and microscopically according to the situation of the diphtheritic affection. On those mucous membranes which are covered with a smooth, stratified epithelium (as mouth, nose, pharynx, vagina), the exudation is, generally speaking, firm and tough. The irregularly distributed exudation-mass is mingled with epithelial cells and pus cells. Where the diphtheritic exudation is closely connected with the mucous membrane, there are found enormous masses of articulated and branched thallus-threads.

"On those mucous membranes which are covered with cylindrical or ciliated epithelium (larynx, upper part of trachea, intestine), the diphtheritic exudation has a softer and more creamy consistency, so that it can be easily stripped off with the back of a knife or with forceps. The epithelial cells are completely consumed, and portions of the exudation appear under the microscope as masses of detritus pierced with fungus filaments.

"Diphtheria and croup may very often coexist and pass the one into the other without being identical.

"Croup may be produced by the inflammatory stimulus supplied by the diphtheritic affection, but may be also a distinct disease existing simultaneously and independently." Letzerich has often observed the fact that the diphtheritic fungus when widely distributed in the tissues (of the larynx) caused an inflammation of connective tissue, which, when propagated to the trachea, produced croup. Post-mortem examination of laryngeal diphtheria in children have often shown croup and diphtheria present simultaneously."

With respect to the parasitic distinction between croup and diphtheria, it is to be noted:

1. That usually these fungi have been found only in cases of undoubted diphtheria, and their distribution through the organism has been thought to explain the constitutional character of the disease.

2. That nevertheless, in some cases (according to Wagner), primary laryngeal croup has been found to be accompanied by such organisms.

It would appear then premature to take the presence of fungi, even if they are assumed to be the cause of the disease, as a sufficient mark of difference between croup and diphtheria.

Summary.—1. It would seem then that the experience of German pathologists supplies no certain anatomical distinction between the two diseases; but, by taking croup as a purely anatomical term, a sufficiently precise meaning can be given to it. In this sense croup is very frequently an accompaniment of, or more strictly, one of the manifestations of the disease diphtheria.

2. In this sense croup is very nearly always, if not invariably, confined to the air-passages, and one of the highest authorities, Rindfleisch, in his last edition ('*Pathologische Gewebelehre*,' p. 307, 5th edit., 1878), clearly shows that the anatomical distinction between croup and diphtheria in the older (German) sense of the words, "obviously depends upon the anatomy of the parts affected." In other words, the croupous process belongs to the air-passages, the diphtheritic to the pharynx and fauces, whatever their causes respectively may be.

3. The presence or absence of vegetable growth cannot be

taken as a distinction between croup and diphtheria, till there are a larger number of negative observations showing the absence of this growth in cases having the clinical characters of croup.

4. Croup, if it exists as a distinct disease, must be defined both by exhibiting the anatomical characters of croup only, without those of diphtheria (infiltration, necrosis, micrococci?) and further by clinical characters, with which here we are not concerned.

APPENDIX IV.

REPORT ON THE HISTOLOGY OF THE LARYNGEAL AND TRACHEAL FALSE MEMBRANE.

IN reporting upon the microscopic examination of the larynx and trachea in cases of membranous exudation in that position we have to regret that the means at our disposal have been very limited, and that, therefore, no decisive result can be expected.

We had hoped to have been able to examine the condition of the air passages, and to have compared the false membrane and the subjacent tissues in various forms.

The conditions which we desired to examine were as follows :

1. Membranous exudation resulting from mechanical, chemical, or physical irritants—boiling water—some cases of which have been recorded, and of which one of us has seen an instance.

2. Membranous exudation limited to the larynx, in which the surroundings, the absence of probable contagion, the history of onset, symptoms during life, and post-mortem appearances, should have excluded, as far as might be, the possibility of contagion.

3. Cases of undoubted diphtheria with laryngo-tracheal exudation, both with and without false membrane in the pharynx, &c.

4. Membranous exudation in the larynx occurring in the course of fever, septicæmia, and the like.

We regret that we have not met with the help we anticipated and hoped to receive in the matter, and that, with two or three exceptions, we have not received any such specimens for examination in spite of repeated requests. We have, therefore, been obliged to rely solely upon the cases which have fallen under our own observation, and as these have been taken from hospital practice it has not been possible thoroughly to exclude diphtheritic contagion. The only exceptions are a specimen kindly forwarded by Dr. Wm. Squire, of which the particulars of the clinical history have been published in the 'Trans. Path. Soc.,' vol. xxvii, and two specimens of the larynx, &c., from cases of membranous exudation following measles, which were under the care of Dr. Dickinson. We have, however, examined specimens

from a number of cases of "diphtheria" and "croup" occurring in hospital practice. In some of these there was distinct evidence of contagion of diphtheria; in others this could not be ascertained. In two of the cases of membranous exudation in the fauces and larynx no source of contagion could be discovered, and there were strong grounds for a belief that they originated in one case from insanitary ward conditions, in the other from the patient's own condition, both being in patients who had been in the hospital for some considerable time. But having in view the many possibilities of importation of contagion from without existing in every large hospital we cannot speak positively on the subject. Three of the cases from which specimens were taken conformed clinically to the type of "croup," and there was no discoverable source of contagion. The only cases which we have been able to examine of false membrane occurring in specific fevers and blood-poisoning are four: one case of typhoid fever in which the false membrane precisely corresponded with that seen in diphtheria, the other a case of septicæmia in which a thin pellicular exudation was found in the larynx only, and two cases following measles. It is clear that any examination of these specimens could not solve the question of the distinction of "croup" and "diphtheria," for we have failed to obtain any specimen from a case to which we could certainly give the former name in its commonly accepted signification; but we believe that the record of the examination may throw some light upon the questions at issue, for the following reasons:

Whatever may be the etiology of the cases in which there is laryngeal membranous exudation only, the result of our observation is to lead to a belief that such exudation may occur under conditions which negative the probability of the presence of contagion. The similarity of the appearance, relations and mode of formation of the false membrane under the various conditions in which it is found, lead to the belief that if "diphtheria" is a distinct and well-defined specific disease the power of production of false membrane in the larynx and trachea is not peculiar to it, but that its poison shares the power of giving rise to it with other poisons and irritants, and that its distinctive characters must be sought, not in the presence of false membrane, but either in some peculiar characters of the membrane, or in other and independent conditions. In fact, it appears to us that the formation of false membrane in the larynx and trachea is merely a mode of reaction of the mucous membrane which may be set up by a variety of conditions.

In the examination of the larynx and trachea with the false membrane we have endeavoured to determine the following points:

Are there any distinctions in the mode of formation of the false membrane and its general characters, and in its relations

to the subjacent tissues, such as have been stated to exist and to be distinctive of "diphtheria" and "croup?"

Does the false membrane contain any "specific" elements, &c., such as bacteria, fungoid growths, and the like, in any cases, and do the cases in which such elements are found coincide with any special class of symptoms or special etiology?

In the decision of these questions we have kept in view the changes—1, in the membrane itself; 2, in the mucous and sub-mucous tissues; and 3, in the deeper parts and surrounding structures.

We have examined the parts in the fresh condition by means of teasing and with various reagents, and also sections made after hardening in absolute alcohol, in methylated spirit, and in Müller's fluid, and stained with various reagents or unstained.

We give only a brief summary of the results which we have obtained, believing that a minute description of all the appearances, apart from drawings, would be unintelligible and valueless, and that a minute description of each case would equally be out of place.

False membrane.—When examined in a fresh condition by means of dilaceration in water or glycerine this appears to consist mainly of altered cells, many of which in size and shape resembled ordinary pus-corpuscles, others of more angular shape and larger size, and yet others large, granular, with two or three nuclei, and some scarcely altered columnar epithelial cells, with others more or less swollen and granular. Together with this some fibrils and a good deal of granular matter were observed. In one case groups of minute granules resembling micrococci were seen, but in others there was no such appearance. It should be added that in only some of the cases was the membrane examined in this way.

By sections made through the membrane *in situ* with the subjacent structures, either fresh or after various processes of hardening and staining, &c.

By this method of examination certain differences were observed in different cases, and in different parts from the same case. The false membrane formed a pretty continuous layer, usually closely adherent to the subjacent tissues or only separated here and there, and at its margin terminating abruptly. The false membrane, as examined with a low power (one inch), appeared in some cases to be composed of two layers, not separate, but marked by a somewhat wavy line, which was somewhat darker than the rest when stained, and seemed to indicate a transition in the structure, but not a separation. Where these two layers existed they will be spoken of as superficial and deep respectively. The condition of the free surface varied. In some specimens it was well defined and abrupt, but usually it was covered by an irregular layer of altered cells, with some

nearly unaltered epithelial cells, embedded in a granular matrix. This layer passed insensibly into the subjacent membrane.

The "superficial" layers of the membrane appeared for the most part to consist of a sort of coralline structure, irregularly stratified, the strata lying more or less parallel to the surface, the structure appearing under some conditions to be formed by a network with thick meshes and small apertures pretty regularly disposed through it. This network consisted of an apparently homogeneous, highly refractile substance, in which no trace of fibrillation could be seen. In the spaces were usually seen one or two nuclei. But in staining with various reagents and mounting in different ways this appearance was found to be a deceptive one, and it could be clearly seen that the apparent network was made up of cells, fused together at their edges, and having undergone such changes that the nuclei and centres of the cells were readily removed, leaving the marginal portions adherent. The transitional stages of this condition could be readily seen by comparison of sections prepared in various ways. Here and there the bands of the homogeneous network thus formed were thicker and more distinct.

The "deeper" layer, where distinctly present, appeared to consist of a more irregularly arranged network, composed of irregularly interlacing fibrillæ. At its superficial part it passed almost insensibly into the superficial layer, the coralline structure becoming replaced by irregular fibrils, the transition zone being seen best with a low power. Examined with the higher power, this deeper layer was found to be composed almost entirely of an irregularly interlacing network resembling coagulated fibrin, with some leucocytes embedded in its meshes.

In some parts the fibrous bands passed into the gland ducts for some distance, but not constantly.

The connection of the false membrane with the mucous membrane varied in different parts. In some cases a portion of the epithelium remained beneath the false membrane, which lay upon it; this especially when only the homogenous network was present. In other cases the basement membrane only persisted, and to this the fibrillated exudation closely adhered, or was separated at points corresponding apparently with the ducts of glands, the space being filled with fluid containing some altered epithelial cells. Only in some parts, here and there, did the superficial layers of the mucous membrane appear distinctly destroyed, the false membrane penetrating more deeply, and this in no case where the membrane could be readily detached as a pellicle.

The thickness of the deeper fibrinous layers varied in different cases and in different parts of the same larynx, in some being scarcely perceptible.

The deeper structures of the mucous and submucous tissues

presented various degrees of ordinary inflammation, the vessels being distended and filled with blood, and their walls and the surrounding tissue more or less infiltrated with leucocytes. This condition was either limited to the mucous membrane itself, or extended deeply through all the tissues, and in some cases involved the whole of the tissues around (*e. g.* muscles of larynx, &c.). The thickness of the deeper layers of the false membrane and the extension into the gland ducts appeared to be in proportion to the intensity of the inflammation.

The mucous racemose glands in some cases showed but little alteration, or only a slight degree of catarrhal change, but in others they were filled with exudation and catarrhal products, and processes of fibrinous exudation extended into them.

So far, then, we have described the general appearances in all the cases, some in a greater or less degree. We must now define and limit the application to sets of cases.

In all the cases of true diphtheria where the larynx was affected, or where a clear history of contagion was present, and the larynx and trachea were alone affected, the inflammatory changes were very marked in proportion to the amount of exudation, with two exceptions, in which the exudation was very abundant on the posterior nares, fauces, and soft palate, but only commencing in the larynx. But even in these cases the deeper structures (*i. e.* away from the mucous membrane) showed traces of a very intense inflammatory process. The same must be said of one case in which the contagion was doubtful, but the pharyngeal affection was extensive. In these cases the deeper fibrinous exudation was very pronounced and firmly adherent, penetrating into the mucous glands, and the inflammation of the mucous and submucous tissues disproportionately intense.

On the other hand, in the cases in which the laryngeal exudation was the main factor, the lymphatic glands free from enlargement, and the case clinically conformable to "croup," the fibrinous part of the exudation was relatively slight, and the submucous inflammation much less intense or almost absent.

In two cases we were able to study the mode of formation of the false membrane in its early stages. The process appeared to begin as one of multiplication and heaping up of the epithelial cells at certain points, the regular arrangement of columnar cells being replaced by a mass of cells of smaller size and irregularly arranged, this appearing to be the first step in the morbid process.

From a comparative study of cases it would appear that the more superficial layers are formed by a change in the epithelium, consisting in proliferation, adhesion of the cells, either by a change in their substance or by mere exudation between them, and an alteration in the central portion of the cell, the change being very much like that seen in the growth of some vegetable tissues.

After a continuance of this process for a certain time ordinary fibrinous exudation ("coagulable lymph," liquor sanguinis, and leucocytes) appears to be poured out on the surface of the membrane, and, coagulating, becomes adherent to the superficial layers on the one hand, and the basement membrane on the other, and entering into and filling up the ducts of the mucous glands. (That it is *not* exuded from the mucous glands would seem certain, for the epithelium is often unaltered, and the fibrinous plug only penetrates a certain distance.)

When the false membrane is separating it would seem that this is effected either by secretion from the mucous gland raising it up, or by breaking down of the false membrane itself.

An explanation of this change which may be suggested is that the fibrinous exudation does not occur until the epithelium has been more or less detached, leaving only the basement membrane with dilated vessels beneath it, the state being that seen when the epithelium or epidermis is just peeled off (as may be seen in abrasions of the cuticle), an exudation of plastic lymph then occurring.

We have, however, as stated above, seen the epithelium persisting in patches beneath the false membranes (and M. Charcot has recently described a similar appearance).

Special conditions.—The presence of fungi and bacteria or micrococci has been so frequently described as a constant factor in diphtheria that some importance may be attached to the question of their discovery. Our opportunities of observation on this point have been limited, for we exclude from consideration such cases as were only examined some time after death, and in hot weather, or when decomposition had clearly set in; and we should attach but little importance to discovery of micrococci under such conditions. But it may be a fact of importance that in some cases even under such favorable conditions for their development, and in cases of undoubted diphtheria, we have failed to find either micrococci or fungus, except on the exposed surface of the false membrane.

In only one case (viz. a case where contagion was doubtful) have we found undoubted evidence of the presence of the minute ovoid or rounded bodies described by Oertel and others as the peculiar element of diphtheria. In that case we found clusters of micrococci both in the false membrane, in the mucous and submucous tissues, and in the lymphatic spaces of the connective and intermuscular planes, deeply in the substance of the larynx. It must be understood that this statement refers solely to the respiratory tract, not to the pharynx or nares, in which the presence of such minute granules, and even of filamentous network, simulating, if not really such, mycelium, are much more frequently found.

We are unable, therefore, to draw any conclusion from this

point. Of the condition of the pharynx, &c., we have said nothing, as it scarcely belongs to the part of the subject which we have been specially investigating. But we may state that one of us has found in three cases of diphtheria where there was some exudation in the pharynx, a very marked and peculiar swelling of the solitary follicles in the sub-mucous tissue, in some cases quite disproportionate to the amount of exudation, and, so far as could be judged, in some cases preceding the local exudation. These glands become greatly swollen, the elements multiplying or being infiltrated with leucocytes, and rapidly forming a homogeneous granular mass by degeneration of the inflammatory products. In the same way we have found the lymphatic glands of the neck intensely inflamed, with small spots of suppuration in them, and occasionally small hæmorrhages in and around them. But we have not entered here into the study of the changes in the pharynx, &c., which occur in diphtheria, as they appear to be specially excluded by the terms of the problem before us.

The state of the *lymphatic glands* of the neck. Our opportunities for the examination of these were so few that we prefer not to make any decided statement as to their condition.

(In conclusion, we may refer to the observations made twenty years ago by Dr. Bristowe, see 'Path. Trans.,' vol. x, p. 321, and to the description given by Wagner ('Gen. Pathology,' p. 264, et seq.), with which our own observations closely correspond.)

NOTE.—The following case has been communicated to the Committee since the completion of their report, but it has been thought of so great importance that they have appended it, together with a report on the microscopic structure of the cast by the members of the Committee entrusted with that branch of the inquiry. They desire to acknowledge their obligations to Dr. Whitehead Reid for his ready permission to examine the specimen, and to Mr. T. D. Acland, B.A., who has kindly prepared the sections.

A CASE OF MEMBRANOUS LARYNGITIS FROM EAU DE COLOGNE.

Mrs. W. V. L.—, æt. 27 years, having her usual health, received, during a faint from pain at stool, some eau de Cologne into her trachea by way of the left nostril, her head being at the time in a dependent position. Burning and pain were at once experienced in the nostrils, throat, larynx, eyes, and ears, breathing and deglutition became difficult and painful, the fauces and conjunc-

tivæ red, swollen, and congested, and the voice a mere whisper; there was a hoarse, constant cough. Suddenly, within an hour, the extreme dryness of parts changed to profuse watery discharge from eyes, nose, and mouth, producing great relief to the previous strangling sensations. There was no fever, but a burning feeling in the ears and intense conjunctivitis, a constant desire to swallow, wheezy respiration and loose cough, with mucous expectoration. Several spasmodic exacerbations of dyspnoea occurred during the day. Pulse 100 to 120.

During second day, which was her worst, the cough became more incessant and "croupy" in character. Deglutition became so extremely painful that nutrient enemata were resorted to. The respiratory murmur became feeble, there were loud mucous râles in the trachea, and symptoms of defective oxygenation. By the evening she was greatly depressed. Pulse 130, feeble; cough incessant; great dyspnoea and orthopnoea; rhonchus in both lungs. Two superficial abrasions, with irregular edges and raw surfaces, were noticed on the upper part of the pharynx. There were no other excoriations nor ulcers seen in the mouth. Propped up in bed a very restless night was passed, the breathing being stridulous throughout.

On the third morning the ulcers were covered with membrane; a little bright blood was coughed up, followed by a piece of soft elastic membrane. The cough was constant and "brassy;" expectoration thick, white, and tenacious, and sank to the bottom of water; voice aphonic; breathing audible beyond the room; respirations shallow and rapid. Examination of the chest proved the left bronchus to be most obstructed. Skin was moist; temperature 99.4°; bowels loose; pulse 100, feeble. She had no sense of taste nor smell, but was not deaf. There was photophobia, lachrymation, and congestion of the lower palpebral conjunctiva. Tongue moist, furred white, with prominent papillæ and red edges. A thin, glairy, and irritating secretion ran from the nose; the edges of the alæ internally were excoriated and swollen, more especially in the left nostril. Pillars of fauces were dusky; tonsils scarcely apparent and not affected; back of pharynx was bright red, and a thick, yellowish pellet was hanging down behind the soft palate to the right side, covering the site of the two ulcers. Soft palate was congested and uvula somewhat elongated. The whole upper part of the pharynx was inflamed. The epiglottis scarlet, a few vessels apparent on it. There was some oedema of the glottis, especially of the mucous membrane about the arytenoids, which was thickened, and of the aryepiglottic folds and ventricular bands; it was difficult to recognise the vocal cords, they were congested posteriorly at least, and some tenacious secretion occupied the glottis. The laryngoscopic examination was ill-borne and rhinoscopy found impossible. There were no enlarged glands.

On the fourth day the cough was more urgent, with a constant desire to expel something; the œdema of the glottis was greatly reduced by treatment, and a small piece of membrane was again brought up in the evening.

The cough became more violent during the night, and on the fifth day, at 4 a.m. (92 hours from the accident), a perfect "cast" of the larynx, trachea, and upper part of the left bronchus was expelled entire, in one piece, with immediate and great relief, her voice returning at once. The "cast" measured six inches in length, and (after having been exposed for eleven hours on a handkerchief in a hot room) would admit an ordinary cedar pencil down its lumen. It was yellowish-white in colour, very thick, soft, elastic, and full of moisture. The impressions of the cricoid cartilage and ten rings of the trachea could be distinctly counted upon its surface; its upper extremity was frayed out, and its lower filiform, though hollow. It was stained with bright blood at its upper part, at the filiform extremity, and midway between these points. A little bright blood was brought up after the expulsion of the cast. In the evening her pulse was 88, fuller, and temperature 97.4° .

The œdema of the glottis had now quite disappeared. The larynx remained tender for six more days; the vocal cords were dull and congested, but never ulcerated; they did not regain their lustre till after the twelfth day.

Small pieces of membrane were coughed up to the end of the seventh day, on which day there was some slight return of taste, the tongue peeling in patches, leaving raw, red-looking place amid the white fur. The left tonsil also became painful, swollen and red on this seventh day, and there was slight glandular enlargement at the angle of the jaw then, but this quite passed off by the end of the ninth day. There was never any ulceration of, nor any patches on, the tonsil; the right one was not at all affected. The left Eustachian tube was found to be blocked at the time the tonsil was inflamed, but air entered the right tube freely. The left ear was also painful on swallowing and yawning, and both nostrils were impervious at this time. Small patches of membrane remained in the left nostril up to the seventh evening. The nostrils remained blocked till after the ninth day. The mucous membrane of the nose had returned to its natural state by the eleventh day. Lactic acid and lime-water visibly dissolved the patches of membrane in the left nostril.

From the fifth to the twelfth day the patient suffered from indigestion and constipation, and passed mucous shreds in abundance when the bowels were made to act. The urine was very acid, loaded with amorphous lithates and epithelium (not renal); sp. gr. was 1036. It never contained albumen. The catamenia, which were due at this time, did not appear. By the thirteenth day some sense of smell returned.

The cough continued till after the fourteenth day; the eyes remained sensitive to light up to the twentieth day; a disagreeable odour at the back of her nose annoyed her also then.

In three weeks from the accident she could sing again. There were never any paralyses. Her recovery was perfect with the exception of some diminution of acuteness of her gustatory and olfactory sensations. Three months afterwards she said, "Things frequently smell and taste of Eau de Cologne." She was exposed to neither scarlatinal nor typhoid poisons either before or during her illness. There was no diphtheria in the village, and neither of her young children, who were constantly with her, became ill.

T. WHITEHEAD REID,
Surgeon to the Kent and Canterbury Hospital.

Report on the microscopic characters of the cast of trachea produced by Eau de Cologne.—Four sections were submitted for examination. They formed perfect casts of the interior of the trachea. All of them presented nearly the same characters. The outer layers present a lamellated appearance, studded with fine nuclei. These lamellæ are mostly in close contact with each other. More internally the texture of the cast becomes looser, and the lamellæ are separated by spaces from each other, the spaces containing a granular coagulum. Still more internally the lamellæ become again closely united. In parts, more especially towards the lumen of the tube (the internal layers), altered epithelial elements are visible, each cell preserving more or less its original cylinder form, and the whole forming in the cast a layer traceable for some distance, but never traceable completely round the tube. Nor does this epithelial layer abut anywhere directly upon the internal surface of the tube, being always separated from it by more or less granular coagulum, apparently formed by transudation from the cells before these were cast off themselves in the form of a definite membrane. The middle looser layers of the cast present many of the appearances of mucinous coagulum. Here and there the spaces contained between the lamellæ are of a circular or oval outline, and appear to represent, or to be casts of, gland tubes. In one or two, these spaces are lined with conical or triangular cells, which are evidently the altered epithelial linings of these ducts. Comparing the nuclear proliferation in the inner layers with that in the outer layers, we find that the nuclei are more abundant, more closely packed, and connected together by more definite intercellular tissue in the outer layers than in the inner.

From the above it would appear as if the cast had been produced in the following manner and in the following layers:—
(1) A layer of muco-albuminous coagulum lying on the inner

surface of the cylindrical epithelium, and possibly produced by the direct action of the eau de Cologne on the epithelium. (2) A layer of altered cylindrical epithelial cells, arranged so as to resemble papillæ. The papillary arrangement must, however, be due to the normal epithelium being pushed off into the interior of the tube by the exudation of a considerable quantity of material on the side of its attached surface, and thus being thrown into plaits, which in section simulate the form of papillæ. This epithelial layer would appear to have been dislodged from its underlying basement membrane by—(3) A thick layer of mucosalbumen, loosely coagulated in a lamelliform manner, containing scattered nuclei and enclosing spaces, which appear to be partly casts of ducts and partly mere intervals between the lamellæ of coagulated mucin. (4) The last layer is backed up externally by a more definite layer of nucleated albuminous tissue, which appears to result from the direct proliferation of the nuclei, &c., of the tracheal wall.

Comparing this cast with the diphtheritic membrane in the section of the larynx from one of Dr. Dickinson's cases following measles, and another of ordinary diphtheria, prepared by Dr. Greenfield, it is evident that the only part in the diphtheria sections strictly comparable to the above is the layer in the latter, numbered 4. We must, however, guard against concluding from this that there is never produced in diphtheria anything comparable to the layers 1, 2, and 3, because the one is a section of the larynx with membrane adhering to it, whereas the other is a mere section of a cast thrown off during life.

2701

APPENDIX V.

ANALYSIS OF CASES OF LARYNGITIS WITHOUT EVIDENCE OF MEMBRANE (NON-MEMBRANOUS CROUP), OF MEMBRANOUS LARYNGITIS, AND OF PHARYNGEAL DIPHTHERIA, IN DR. DICKINSON'S PRACTICE, MOSTLY AT ST. GEORGE'S HOSPITAL AND THE HOSPITAL FOR SICK CHILDREN.

TABLE I.—(A) *Non-membranous laryngitis.*

Reference.	Name.	Age.	Month of attack.	Cause.	Preceding symptoms and onset.
Hosp. for Sick Children	Jessie Sutton	15 mos.	Nov.	—	Cough 3 weeks; sores on tongue; blebs on lips; whooping inspiration 10 days before death; dyspnoea and recession

(B) *Non-membranous laryngitis. Presumptive*

Ditto	Thomas Woods	2 yrs. 4 mos.	Aug.	—	Croupy attack not very severe; recession; dyspnoea
	(2nd attack	3 yrs.	May		Cough for 1 day; at night croupy
Ditto	James Chaplin	10 yrs.	April	Walked to Woolwich on cold, windy day	Lost voice when he got there; breath at once short; croupy cough, &c., 3 days later
Ditto	C. Lacon (M.)	13 mos.	Nov.	—	Running at nose and eyes; next night croupy cough; much stridor; dyspnoea severe; much recession of chest
Ditto	Fred. Rader	1 year 6 mos.	March	—	Puffy across nose, but no running laryngeal cough; some recession
Ditto	Alfred Peters	4 yrs.	Aug.	—	Croupy cough came on at 2 a.m.
Vol. ii, p. 172	Charles Renn	5½ mos.	Feb.	Subject to bronchitis; no cause recognised	Woke up in night with cough, wheezing, and intense dyspnoea of laryngeal character. (Fell on head 2 days before attack.)
Vol. xvii, p. 308	L. W. (M.)	10 yrs.	Jan.	Had been on the previous day to the Crystal Palace with a footman	Suddenly seized in night with croupy symptoms; extreme laryngeal dyspnoea; tracheotomy is imminent
Hosp. for Sick Children	Robert Sacoman	5 yrs.	Nov.	—	Marked croupy symptoms followed upon running at nose and eyes and cough of a fortnight's duration; measles came out on 4th day after croup
Ditto	Robert Whalley	4 yrs.	Dec.	—	Cough 2 days before rash of measles; next day laryngeal dyspnoea as croup complete; rash came on and explained it

Positive evidence of absence of membrane.

Membrane as observed in life.	Albu-minuria.	Treatment.	Re-sult.	Post-mortem.	Remarks.	Date of trachy. from 1st lar. or phar. sympt.	Dura. of dis. from 1st lar. or phar. sym. to rec., death, or removal.
—	—	Anti-mony, emeti-cally and otherwise	D.	Edema of larynx and 2 or 3 upper rings of trachea	Died at home rather suddenly had been taken out on suspicion of whooping cough	—	Hoarse 4 weeks; laryngeal dyspnoea 9 days.

evidence of absence of membrane.

None seen	—	Anti-mony	R.	—	Running from nose; swollen gland on each side of neck	—	14 days.
None seen; tonsils red	—	—	R.	—	—	—	7 days.
None seen; pharynx red	—	Anti-mony	R.	—	—	—	85 days.
None seen; fauces congested	—	Anti-mony and Ipecacu-anha	R.	—	Inflammation of conjunctiva	—	25 days.
None seen; pharynx natural	—	Leeches and anti-mony	R.	—	—	—	13 days.
None seen; tonsils large, red, and showed spots of secretion	—	—	R.	—	Enlarged glands on either side of jaw	—	14 days.
None either seen in throat or coughed up	—	Anti-mony	R.	—	—	—	11 days.
None seen	—	Ditto	R.	—	Three previous attacks; weather on last cold and windy	—	About 1 day.
Ditto	—	Ditto	Sent out	—	Croup in these cases; early symptoms of measles	—	3 days.
Ditto	—	Carbolic spray	Do.	—		—	3 days.

Reference.	Name.	Age.	Month of attack.	Cause.	Preceding symptoms and onset.
St. George's	Daniel O'Connell	3½ yrs.	Nov.	—	Went to bed well; awoke with cough and dyspnoea, which became extreme, and was accompanied with bronchitis; improved for a time, then relapsed, and was taken out in a state of much dyspnoea. (Possibly membrane present, though none seen)
Hosp. for Sick Children	Cornelius Leary	3 mos.	May	—	Subject to cough; cough increased and became croupy
Ditto	Alice Didman	7 yrs.	May	Lay on cold stones in yard	Sore throat for 2 days; croupy dyspnoea came on suddenly at 1 a.m.; croupy symptoms severe and protracted
Ditto	Mary A. Blythe	8 yrs.	June	Caught cold 14 days before admission	Tracheotomy on point of being performed
Ditto	Wm. Harrison	1 year 3 mos.	March	—	Loss of appetite, feverishness, cough, croup
Ditto	Stephan Hagan	1 year 6 mos.	Dec.	—	Hoarseness, cough; woke up suddenly with croupy dyspnoea at 1 a.m.
Ditto	Lawrence Donovan	3 yrs. 7 mos.	Dec.	—	Cough 3 months; spat blood 3 days before attack; attacked suddenly at 7.30 a.m. (woke up); running at eyes and nose on admission
St. George's	Bridget Cullen	6 yrs.	May	Went to bed apparently well, but for a slight cold evening before	Woke at 3 a.m. in a fit of dyspnoea, choking, and making a great noise; mother thought she would have died; brought to hospital 3 hours later in little dyspnoea, but with hoarse cough

TABLE II.—*Membranous laryngitis. Membrane, as far as*

Hosp. for Sick Children	Albert Abbott	10 mos.	Feb.	—	Preceded by 7 days' diarrhoea; brother had stomatitis; begun with gradual accession of croupy breathing
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MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX V. 105

Membrane as observed in life.	Albu- minuria.	Treatment.	Re- sult.	Post-mortem.	Remarks.	Date of trachy. from 1st lar. or phar. sympt.	Dura. of dis- from 1st lar. or phar. sym- to rec. death, or removal.
None seen (but suspected)	Alb., at first a trace afterwards much	Anti- mony	Ta- ken out	—	Membrane pro- bably present in this case?	—	15 days.
None seen	—	—	Do.	—	—	—	3 days.
None seen; brought up thick mucus	No alb., trace of sugar	Anti- mony	R.	—	Rose-rash with- out rise of tem- perature on 5th day	—	Croupy dyspnœa 1 month; sore throat 3 days before.
Fauces normal; none evacuated	No alb.	Emetics, antimony	R.	—	Had 3 attacks of same sort, the one described being the 2nd	—	44 days.
None seen	—	Ditto	R.	—	—	—	44 days.
Fauces a little swollen; no membrane seen	—	Anti- mony	R.	—	Dyspnœa great; child rickety; case unpromis- ing; much re- cession	—	10 days.
Tonsils red and swollen, but no membrane	—	Ditto	R.	—	—	—	Cough 3 months; croupy seizure 10 days. 11 days.
No membrane; tonsils and throat congested slightly	No alb.	Ipecacu- anha, antimony	R.	—	Symptoms ur- gent, but with intermissions for about 24 hours, then gave place to ordi- nary bronchitis	—	
<i>could be ascertained, confined to larynx and air-passages.</i>							
Pharynx con- gested; thin membrane on larynx; bron- chitis	—	Anti- mony	D.	Thin membrane on larynx only, pharynx and bronchial tubes congested	—	—	2 days.

106 REPORT OF THE COMMITTEE ON THE RELATIONS OF

Reference.	Name.	Age.	Month of attack.	Cause.	Preceding symptoms and onset.
Hosp. for Sick Children	James Cull	4 yrs.	April	—	Croupy cough succeeded by croupy breathing, 2 days
Ditto	John Dowden	2 yrs.	June	—	Feverish for 6 days, then breathing became difficult and noisy
Ditto	Walter Conquest	5 yrs.	June	Want of air and food; 6 people living in one room	Headache, stomachache, and feverishness; had a cough, got choky, and complained of throat; no running at nose
Ditto	Alfred Churchill	16 mos.	July	—	Running at nose for a week; cough, which became croupy
Ditto	Marriner Hutson (F.)	6 yrs.	Feb.	Succeeded upon typhoid; escape of sewer gas into ward	Pneumonic signs during typhoid convalescence; these succeeded by ringing cough and the expectoration of tubes of membrane
Ditto	James Smith	4 yrs.	Feb.	Succeeded upon measles	Delicate, diarrhoea, &c., caught measles in hospital; hoarse (croupy) cough 2 days after appearance of rash; died within 24 hours of recognition of tracheitis
Ditto	Herbert Haley	2½ yrs.	March	Ditto	Large head; otorrhoea; caught measles in hospital; paroxysms of dyspnoea 2 days after rash, with hoarse cough; died 3 days later more from pulmonary than laryngeal obstruction
Ditto	John P. Lambert	3 yrs.	Feb.	Ditto	In hospital for diabetes insipidus; there caught measles; became hoarse 8 days after appearance of rash; one day out before death

MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX V. 107

Membrane as observed in life.	Albuminuria.	Treatment.	Result.	Post-mortem.	Remarks.	Date of trachea, from 1st lar. or phar. symp.	Date of dis. from 1st lar. or phar. sym. to rec. death, or removal.
None seen in throat, though some expectorated through tube	None	Tracheotomy	R.	—	—	On 3rd	24 days.
None seen in throat; found after death	—	Antimony, tracheotomy	D.	False membrane in larynx and trachea; none above	—	On 2nd	2 days.
No exudations in throat, nor any swelling	Little alb., trace of sugar	Antimony	D.	Membrane confined to air-passages from epiglottis downwards; bronchitis	—	On 5th	5 days.
Nothing on tonsils; no membrane seen in throat, but some expelled from wound	—	Tracheotomy	D.	Thin membrane from true cords to primary bronchi	—	On 2nd	3 days.
Throat perfectly natural to examination, but membrane repeatedly coughed up; tracheotomy	Alb. after operation	Antimony, carbolic spray, tracheotomy	R.	—	At same time another child had in same ward pharyngeal diphtheria, and a nurse follicular tonsillitis; child's (Hutson's) bed between window and door, but no further reason to attribute attack to cold	On 6th	35 days.
"Fur" on tonsils like that on tongue, but no membrane	No alb.	—	D.	Larynx, trachea, and large bronchi lined with a thick membranous sheath, none on fauces	—	—	3 days.
Fauces clear; glands behind jaw enlarged	—	Antimony, carbolic spray	D.	Much membrane in larynx nearly closing glottis; none in fauces or in trachea or bronchi; lobular pneumonia	—	—	3 days.
No membrane seen during life	—	Quinine, &c.	D.	Larynx and trachea coated with lymph; none in fauces; hepat. in left lung	—	—	1 day.

TABLE III.—*Membranous laryngitis, together with slight*

Reference.	Name.	Age.	Month of attack.	Cause.	Preceding symptoms and onset.
St. George's	Jane Suckling	8 yrs.	June	Has had about a dozen previous attacks of "croup"	Soreness and stiffness about larynx; no nasal discharge or glandular swelling
Hosp. for Sick Children	Annie Thorp	15 mos.	Dec.	No bad smells	Langour, feverishness; after 12 hours quickened breathing, then stridor
Ditto	John Wells	2 yrs. 3 mos.	Nov.	Two brothers and one sister subject to "croup"	Previously well; woke up at 10 p.m. with strange cry; croupy symptoms rapidly supervened
Ditto	Aubrey Peters (M.)	3 yrs. 10 mos.	June	—	Suddenly seized with croupy cough; afterwards laryngeal dyspnoea
Ditto	Ellen Brougham	3 yrs.	May	Sent back from Highgate; "got cold" 12 hours before attack	Formerly had strumous abscesses; no premonitory symptoms noted; stridulous breathing and croupy cough
Ditto	Ellen Smith	8 yrs. 11 mos.	Nov.	—	Slight running at nose, with hoarseness; headache; barking cough; loss of voice and laryngeal dyspnoea
Ditto	William Wood	3 yrs. 3 mos.	May	Cough repeatedly, with running at nose	This succeeded by white spots on tonsils (as stated), and this by gradual accession of laryngeal breathing and stridor
Vol. xvii, p. 189	Maria Sault	3 yrs. 10 mos.	Sept.	—	Fever and slight cough 3 days before stridor

affection, membranous or follicular, of tonsils or fauces.

Membrane as observed in life.	Albuminuria.	Treatment.	Result.	Post-mortem.	Remarks.	Date of trachy from 1st lar. or phar. sympt.	Duration of dis. from 1st lar. to phar. sympt. or death, or removal.
Extensive follicular tonsillitis; no membrane seen in throat, but some seen in trachea	Much	Leeches, anti-mony, tracheotomy	R.	—	Lungs remarkably free from bronchitis. Gave complaint to nurse	On 6th	41 days.
Fauces congested; no membrane seen until after death	—	Anti-mony	D.	Patches of membrane on pharynx, membrane from larynx to 3rd division of bronchi, and leading below that into pus	Much bronchitis	—	2 days.
Small white patch on right tonsil; membranous shred in vomit	No alb.	Anti-mony, tracheotomy	D.	Soft membrane lining whole length of trachea and bronchi	—	On 2nd	3 days.
"Aphthous deposit" on tonsils during life	Slightly alb., afterwards none	Anti-mony, emetics, tracheotomy	D.	Larynx, trachea, and bronchi to 4th divisions lined with exudation	—	On 2nd	4 days.
Nothing seen in throat	Trace = $\frac{1}{10}$, afterwards $\frac{1}{2}$	Ditto	D.	Membrane from larynx to tertiary bronchi; patches on aryteno-epiglottidean folds	—	On 2nd	5 days.
Patch of exudation on tonsil and another on pharynx; membranous ridge within glottis; much membrane vomited and expectorated through tube	No alb. (repeatedly examined)	Ditto	R.	—	—	On 5th	33 days.
Membrane expelled through tube	No alb., later = $\frac{1}{2}$ = $\frac{1}{3}$	Pot. Iod., Liq. Hydrg., Tracheotomy	R ?	—	Intercurrent measles, afterwards œdema; recovered in general health, but never did without tube	?	?
None seen in throat before death	No alb.	Emetics, anti-mony, tracheotomy	D.	Soft membrane in larynx, trachea and bronchi; small patch behind each tonsil	—	On 3rd	4 days.

110 REPORT OF THE COMMITTEE ON THE RELATIONS OF

Reference.	Name.	Age.	Month of attack.	Cause.	Preceding symptoms and onset.
Vol. xvii, p. 192	— Vokes (M.)	2½ yrs.	Sept.	—	Began with croupy cough without preceding illness; dyspnoea became extreme; tracheotomy imminent
St. George's	Amelia Jackson	4 yrs.	Nov.	Supposed to have caught cold	Cough for 4 days before croupy symptoms; sudden dyspnoea in the night; enlarged glands in neck
Hosp. for Sick Children	Frederick Levy	1 year	April	—	Fortnight before attack spots on gums, lips, and tongue; difficulty of swallowing, then of breathing, and sudden attack of croupy symptoms in the night
Ditto	Bridget Edwards	1 year 8 mos.	June	Child held to open window when could not be taken out	Enlarged glands in neck for a week, then stridor and croup
Ditto	George Price	2 yrs.	March	—	Croupy cough; excavation in tonsil with white contents; "suspicious" of diphtheria; afterwards laryngeal dyspnoea and signs of pneumonia

TABLE IV.—*Membrane extensively present*

Vol. xvii, p. 125	Dr. M—'s boy	2 yrs.	June	—	Pale, sickly, and without appetite for 10 days, then membrane seen on fauces; laryngeal dyspnoea several days later; bronchitic sounds
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MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX V. 111

Membrane as observed in life.	Albuminuria.	Treatment.	Result.	Post-mortem.	Remarks.	Date of trachey. from 1st lar. or phar. sympt.	Duration of dis. from 1st lar. or phar. sym. to rec. death, or removal.
Patch of membrane seen in throat (by Dr. Martyn), and nitrate of silver applied	—	Antimony	R.	—	—	—	9 days.
Membrane on both tonsils, some coughed up through tube	—	Antimony, emetics, tracheotomy	D.	Membrane on tonsils, back of pharynx, larynx, trachea, and down to smaller bronchi	—	On 2nd	3 days.
No membrane seen in throat, but fauces and tonsils red	Trace of alb.	Antimony	D.	Membrane on posterior wall of pharynx, top of uvula, epiglottidean folds, larynx and trachea; some patches also in bronchi	—	—	4 days.
Membrane on tonsils; membrane expelled through tube	Drawn from bladder p.m., highly alb., full of casts	Emetic, antimony, tracheotomy	D.	Tonsils ulcerated; membrane on epiglottis (pharyngeal aspect), also larynx and upper part of trachea; below congested	—	On 2nd	3 days.
White matter on tonsil as described; enlarged glands behind jaw	—	Carbolic spray	D.	Membrane in upper part of glottis and on thyroid cartilage; trachea and bronchi congested and full of purulent fluid; hepat. of left lung, which, with the right, contained tubercles	—	—	18 days.
<i>in throat as well as in air-passages.</i>							
Tube of membrane coughed from trachea, beside what was seen on fauces	—	Quinine and iron	D.	—	—	—	From memb. 11 days, laryngeal 1 day.

112 REPORT OF THE COMMITTEE ON THE RELATIONS OF

Reference.	Name.	Age.	Month of attack.	Cause.	Preceding symptoms and onset.
Vol. xvii, p. 238	Caroline Harris	9 mos.	May	—	Sore throat, difficulty of breathing, and swelling of glands about jaws; dyspnoea pharyngeal in tone
Vol. xvii, p. 387	Louisa Bird	3 yrs.	June	—	A month before feverish, languid, lost appetite, and had running at nose; got better in a week; after 3 weeks attacked again, shivered, and became feverish again; had swelling at side of neck, then difficulty of swallowing, huskiness of voice
Hosp. for Sick Children	Emily Burnell	3 yrs. 6 mos.	May	No smell or other obvious cause	Felt sleepy; next day talked thick, and 2 days later became stridulous
Ditto	Elizabeth Saala	6 yrs.	Nov.	Drain smells in house. Another child of same family had diphtheria 3 years ago in this hospital	Throat sore 9 days before tracheotomy; membrane on tonsils and pharynx 6 days later
Ditto	Mary Gorfin	3 yrs.	Jan.	—	Feverish and sick; next day fauces in throat; became "light-headed;" diarrhoea
Ditto	Harriett Lester	4½ yrs.	Aug.	Family lived over a stable, drank from pump just outside	Hoarseness, hoarse cough, at same time feverish, followed by choking dyspnoea in fits, and tracheal respiration
Ditto	Eliza Barnes	7 yrs.	Feb.	Subject to sore throat since scarlatina 3 years before. Attributed to exposure to snow	Caught severe cold in head, chest, and throat; 3 days later breathing became difficult

Membrane as observed in life.	Albuminuria.	Treatment.	Result.	Post-mortem.	Remarks.	Date of trachy. from 1st lar. or phar. sympt.	Duration of dia. from 1st lar. or phar. sym. to rec. death, or removal.
None seen during life	—	Puncture of pharynx, antimony	D.	Membrane behind soft palate and on left tonsil, also in larynx, trachea, and to smallest bronchi; small abscess behind pharynx	Post-pharyngeal abscess suspected, punctured for but not hit	—	9 days.
Membrane on both tonsils and fauces (Nitrate of silver had been applied)	Highly alb.	Caustic before admis., emetics, antimony, tracheotomy	D.	False membrane on both tonsils, and from epiglottis to bronchi of 4th division	Papilliform rash over back, passing into pustules; ulcers at corners of mouth; slight discharge from eyes	On 6th	6 days.
Pimples said to have been seen on throat, brush applied; membrane on tonsils, uvula and pharynx, also within epiglottis, also came out of tube	½ alb., ½ alb.	Tracheotomy	D.	Membrane on soft palate, tonsils, epiglottis, and bronchi	—	On 3rd	6 days.
Membrane on tonsils, and stated to have been seen on pharynx; membrane coughed from wound	Alb. = ½, trace of sugar	Ditto	R.	—	No swelling of glands of neck; mother and another child had sore throats at same time	On 6th	33 days.
Membrane on palate	Urine not obtained	Perchlor. of iron, chlorate of potash	D.	Tonsils, uvula, soft palate, epiglottis, and larynx covered with membrane; membrane not below vocal chords	—	—	6 days.
Exudation on fauces and right tonsil	Trace of alb.	Carb. acid spray and chlorine, iron and quinine	R.	—	—	—	30 days.
Fauces and uvula coated with yellowish white exudation	No urine to be got	Tracheotomy	D.	Extension in pharynx, also in larynx and trachea; not in bronchi	—	On 3rd	4 days.

Reference.	Name.	Age.	Month of attack.	Cause.	Preceding symptoms and onset.
St. George's	Lizzie Bealey	2½ yrs.	Nov.	Followed upon hooping-cough; accompanied scarlatina?	Much glandular enlargement and discharge from nose; much febrile disturbance; said by the doctor to have scarlatina (and had the rash); much coryza; snoring
Vol. xviii, p. 268	W— (F.)	4 yrs.	Jan.	Scarlatina	On same day that rash came out much membrane in throat, which was sponged with nitrate of silver; that evening croupy symptoms, and long tube of tracheal membrane coughed up

TABLE V.—*Membrane on tonsils, fauces, or*

Hosp. for Sick Children	Lavinia Gale	11 yrs.	July	Drank water from butt, which also supplied W.C.?	Pain in swallowing for a few days
Ditto	Edmund Nowell	10 yrs.	Nov.	Water from cistern over W.C.?	Pain in neck behind jaw; liquids returned through nose
Ditto	Ellen Deacott	4 yrs.	Oct.	Came from All Saints' Orphanage	Jaundice 3 weeks before; mode of accession not noted
Ditto	Daniel Rich	1 yr. 7 mos.	Jan.	Smell of drains?	First noticed swelling in neck; child then thirsty and feverish
Ditto	Charles Denley	10 yrs.	Dec.	Vomiting and headache for some weeks before	Languid and ailing, and had sore throat
Ditto	Henry Mills	3 yrs.	Feb.	1 brother now in hospital with diphtheria, of which another died 3 weeks ago	Observed to be poorly in morning in afternoon white patch discovered

Membrane as observed in life.	Albuminuria.	Treatment.	Result.	Post-mortem.	Remarks.	Date of trachy. from 1st lar. or phar. sympt.	Date of dis. from 1st lar. or phar. sym. to rec. death, or removal.
—	—	Tracheotomy	D.	Pharynx, larynx, and upper part of trachea covered with membrane	Probably scarlatinal	On 3rd	4 days.
Much seen on throat, and tracheal tubes coughed up	—	Chlorine and stimulants	D.	—	Several other children had scarlatina same time	—	2 days.

pharynx, to exclusion of air-passages.

Membrane on tonsils, which were swollen and red	Trace	Chlorine and carb. spray, alt. iron and quinine	R.	—	—	—	17 days.
Continuous over back of soft palate, uvula, and tonsils	Ditto	Tinct. of iron to throat, carbolic spray, iron and quinine	R.	—	Breath offensive; enlarged cervical glands	—	25 days.
Opaque membrane on right half of fauces	Ditto	Chlorine and carb. locally, iron, &c.	R.	—	Glands of neck slightly enlarged; memb. left ulcer, which long remained	—	20 days.
Membrane seen all over fauces by Dr. Bridges	—	Iron, quin., pot. chl., car. spray	R.	—	Much swelling of glands in neck	—	8 days.
Patch on right tonsil was scraped off	Alb. $\frac{1}{10}$ to $\frac{1}{20}$	Quinine, iron, pot. chl.	D.	Recent pleurisy; tonsils ulcerated; enlarged suppurating glands about neck	Glands of neck swollen; abscess in neck, which was opened	—	13 days.
White patches on uvula and each tonsil	None	Nit. of silv., car. spray, iron, quin., &c.	R?	—	Ulcer on tongue. Taken out before quite well	—	10 days.

116 REPORT OF THE COMMITTEE ON THE RELATIONS OF

Reference.	Name.	Age.	Month of attack.	Cause.	Preceding symptoms and onset.
Hosp. for Sick Childre	Benjamin Wilson	5 yrs.	July	Attributed to drinking water largely contaminated with dung refuse and drainage (see Analysis). '22 per million of albuminoid ammonia. (Caen Wood, farmer.)	Complained of throat for 5 days
Ditto	John Wilson	7 yrs.	Ditto	Brother of the above. Attacked same time	Feverish, languid, and drowsy; next day throat sore, and had headache
Ditto	Eugene Huon	4 yrs.	June	Child in another part of the house had a croupy cough and recovered	Loss of appetite; looked ill; had lump in neck
St. George's	Ann Hughes (nurse)	29 yrs.	June	In close attendance upon Jane Suckling (Table III)	Throat sore, following evening headache and shivering; no nasal discharge
Ditto	Jessie Goddard	4 yrs.	Dec.	Drain smells in house	Sore-throat, and hoarseness, running at nose

MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX V. 117

Membrane as observed in life.	Albuminuria.	Treatment.	Result.	Post-mortem.	Remarks.	Date of trachey. from 1st lar. or phar. sympt.	Duration of dis. from 1st lar. or phar. sympt. to rec. death, or removal.
Tonsils, fauces, and pharynx swollen and red. On tonsils patches of exudation as large as peas, specks like follicular tonsillitis, or between that and diphtheria	Faint trace	Chlorine and carb. locally, iron and quinine	R.	—	9 children; 1 died a year ago "from diphtheria." 9 days ago mother confined; 2 days afterwards girl, 2 years old, had erysipelas of arm (from slight wound), which spread. She died with sore throat and dyspnoea. (A man and his mother who drank of the same well had violent diarrhoea)	—	29 days.
Tonsils reddened and swollen; no membrane seen; glands at angle of jaw swollen	None	Ditto	R.	—	At same time another child aged rather more, who slept in same room, had sore-throat (diphtheria which extended into larynx, as stated by Mr. Evans, who attended) and died with spasmodic dyspnoea. Baby sickened also and died with purulent ophthalmia and inflammation about navel. All three died within a week. P.S.—Another child in hospital under Dr. Cheadle, belonging to same family, with same symptoms as Benjamin	—	24 days.
Patch of membrane over one of the follicles	Ditto	Carbolic spray, iron, and quinine	R.	—	Large gland below angle of jaw; spleen enlarged	—	5 days.
Uniform white layer over left tonsil, smaller patch on right; sharply defined edge; unconnected with follicles	Ditto	Carbolic spray and chlorine gargle, iron, and quinine	R.	—	—	—	10 days.
Extensive false membrane on palate and fauces	Alb.	Carbolic spray, iron, quinine	D.	Extensive membrane on palate and left tonsil; air-passages free; had running from the nose	—	—	Membrane seen 8 days.

118 REPORT OF THE COMMITTEE ON THE RELATIONS OF

Reference.	Name.	Age.	Month of attack.	Cause.	Preceding symptoms and onset.
Hosp. for Sick Children	Charles Wren	5 yrs.	May	Dung heap under window; also manure pit close by, which was occasionally emptied with much stench. One of the children taken ill day after it was opened	Pain in neck; vomited; shivered; same day mother saw white patch on throat; swelling of glands
Ditto	Lillian Tingey	7 yrs.	April	Water-supply a cistern above a stinking w.c.	Pain in swallowing evening before
Dr. Forshall's case	Dr. P—y	22 yrs.	March	Infection. Attending medically on a nephew 4 years old, who died of diphtheria, making applications to throat, &c. Attacked day of child's death	Feeling of illness and fever; membrane on throat same evening
Mr. Haffenden's case	William W—	45 yrs.	May	All the drainage of the house went into a cesspool a few yards from it, which had not been emptied for more than 15 years. Drinking water from a separate and pure source	Confined to bed with a severe attack of sciatica, which caused much weakness; then complained of difficulty of swallowing and speaking, but had no rigor; membrane then found on soft palate

MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX V. 119

Membrane as observed in life.	Albu-minuria.	Treatment.	Re-sult.	Post-mortem.	Remarks.	Date of trachea from 1st lar. or phar. sympt.	Dura. of dis. from 1st lar. or phar. sym. to rec. death, or removal.
Membrane on both tonsils	Trace of alb.	Carbolic spray, chlorine	R	—	Another child of same family in hospital with pharyngeal diphtheria, same time a third had sore-throat	—	19 days.
Membrane on both tonsils; enlarged glands	Ditto	Ditto	R.	—	3 children in same family died of scarlatina; 3 now patients in hospital with diph.	—	18 days.
On fauces, tonsils, and back of pharynx	—	Carbolic spray, chlorine, tonics	R.	—	The child from whom the disease was apparently caught was one of the subjects of an epidemic then raging. Dr. P. lived in same house and may have been affected from same cause	—	3 weeks.
On soft palate, uvula, and tonsils; lips and tongue ulcerated; little glandular swelling	No alb.	Disinfectants and tonics	R.	—	A child in same house had at same time an attack of well-marked follicular tonsillitis. At some years' interval were two other outbreaks of diphtheria in same house	—	Ditto.

Analysis of Dr. Dickinson's tables. Dates of attack, arranged according to months.

	Class 1.	Class 2.	Class 3.	Class 4.	Class 5.
January	1	0	0	2	1
February	1	4	0	1	2
March	2	1	1	0	0
April	1	1	1	0	1
May	4	0	2	2	2
June	1	2	3	2	2
July	0	1	0	0	3
August	2	0	0	1	0
September	0	0	2	0	0
October	0	0	0	0	1
November	4	0	3	2	1
December	3	0	1	0	2

Description.	No. of cases.	Causes suggested.							Prodromata.						Treatment.				Total results.					
		Exposure to weather (definite).	Foul air or stench.	Foul water.	Infection.	Measles.	Scarlatina.	Abrupt seizure in night	Cough (not measles).	Coryza (not measles).	Sore throat.	Feverishness or rigors. [†]	Malaise.	Diarrhoea.	Albuminuria in urine.	Emetics.	Antimony.	Tonics and anti-sepsis.	Tracheotomy.	Recovered after operation.	Recovered.	Died.	Uncertain.	
1. No membrane seen Of the patients in this series (18 in number), 1 had 2 attacks, 1 had 3 attacks, and 1 had 4 attacks.	19	4	0	0	0	2	0	8	8	3*	1	1	0	0	1‡	4	15	0	0	0	0	14	1	4
2. Membrane confined to air-passages Among these patients (9 in number), the attack was not repeated in any instance.	9	0	2	0	0	3	0	0	4	1	1	2	0	2	2	0	4	3	4	2	2	7	0	
3. Membrane in air-passages, with slight affection of throat One of these patients said to have had "a dozen" previous attacks of croup. She gave pharyngeal diphtheria to her nurse (had also follicular tonsillitis).	13	3?	0	0	0	0	0	3	4	2	3	2	0	6	6	11	1	9	3§	4	9	0		
4. Membrane extensively present in throat as well as in air-passages None of these known to have had similar complaint before; brother of one had diphtheria in hospital.	10	1	1	1?	0	0	2	0	1	2	5	3	1	1	4	1	2	4	5	1	2	8	0	
5. Membrane in throat to exclusion of air-passages None of these patients known to have had similar complaint before. Of the 15 instances recorded in the table there are 8 in which the subject was attacked together with others who were exposed to the same influences. This does not include one case in which while one child had diphtheria another had a croupy cough. The table includes 4 instances in which 3 members of the family were attacked with diphtheria at the same time in close succession, and 3 instances in which 2 persons were similarly attacked with diphtheria or follicular tonsillitis. Among these was one case in which the disease was imparted from a child to her nurse (see Case of Suckling, Table 3), and another in which it was imparted from a child to his doctor.	15	0	4	5	2	0	0	0	0	1	15	6	1	0	8	0	0	15	0	0	12	2	1	
										</														

* Present on admission; date of access not noted.

† Membrane possibly present here.

‡ Independently of measles or scarlatina.

§ One of these, W. Wood, had to wear tube permanently.

Observations upon the foregoing tables. By Dr. DICKINSON.

The first series, *non-membranous laryngitis*, is remarkably distinct. There was no suggestion of zymotic influence in any case; on the other hand, in four the attacks were attributed to cold, in two of which the exposure was fairly definite in nature and result, in both, the patient had been unusually exposed to cold wind during the day, and was suddenly attacked in the ensuing night. It is to be noted that these attacks are apt to begin abruptly in the night, though this mode of commencement is not peculiar to them, inasmuch as it was also noted, though with far less frequency in connection with the membranous affection of the larynx. In a large proportion of the non-membranous class (8 of 19) the attack was preceded by cough; a larger proportion than holds with any form of membranous disease. The presence of coryza furnishes no distinction, as it is occasionally present in all circumstances. The disorder was traced in two instances to incipient measles; in none to scarlatina.

The urine was not found to be albuminous in any case, save in one which it is to be observed was incomplete, and the nature of which was doubtful. The urine was not invariably examined, but it was examined in as large a proportion of instances as in the other classes.

A tendency to recurrence of the attack was strongly marked in this series; one individual having had two attacks, another three attacks, and a third four attacks.

Another distinguishing point is the tendency to recovery or the amenability to treatment. Notwithstanding that dyspnoea in many cases was such as to suggest immediate tracheotomy, yet under steam and antimony, fourteen out of eighteen recovered, one twice, and only one is known to have died. This contrasts very powerfully with all the cases in which laryngeal membrane was known to be present—of the total of which, amounting to 32, 24 ended fatally.

The second class, that of membrane strictly limited to the air-passages, is of small number compared with the instances in which the membranous formation has to a greater or less degree extended above the larynx. It comprises but nine cases. So far as they go they present points of contrast with the preceding class. In no case was exposure to weather apparent as the cause; while in five, the influence of a specific fever or other zymotic poison was indicated, three were associated with measles. In another, a child, Marrener Hutson by name, was seized while in the hospital recovering from typhoid; at the same time that another child in the ward was attacked with well marked pharyngeal diphtheria and a nurse with follicular tonsillitis. The

outbreak was traced to an escape of sewer gas in a water closet opening upon the ward. The child in question, M. Hutson, coughed up a long tube of membrane from the trachea, and eventually recovered after tracheotomy. In the remaining instance the morbid influence was not more definite than the occupation of one room by six persons, of whom the patient was one, together with insufficiency of food. Among these cases feverishness and diarrhœa were noted as precursors of the attack, and in two of the nine, albuminuria as its accompaniment.

But it must be added that, however these cases differed in their progress and result from those of non-membranous inflammation, there was no clinical distinction which could be discerned in the earlier stages.

The third series is that in which membranous affection of the larynx was associated with slight affections of the pharynx or tonsils of the same or a similar kind. In three of these the associated affections took the shape of follicular tonsillitis. This series, together with the one preceding it, may be held to represent what is commonly described as "membranous croup." In the series at present under notice there was a general absence of definitely ascertained cause. In three the attack was hypothetically assigned to cold; in one the child had been habitually held to an open window, but not on any specified occasion with connected result, and in the other instances the action of cold was even more uncertain. Neither in any case was there any definite attribution to foul air or foul water. In the incidence of disease of this class a resemblance to non-membranous croup might be considered to exist in the occasional though less frequent predecession of cough, and in the tendencies each exemplified in one case only, either to attack an individual who has been liable to "croupy" attacks or who belongs to a family, other members of which have had croup presumably of the non-membranous kind. On the other hand, a resemblance to the class of pharyngeal diphtheria could be discerned in the occasional occurrence of premonitory fever. But it must be allowed that neither by what is known of the causes of each attack, nor by the nature of the prodromata, can the class now under discussion be definitely distinguished either from non-membranous croup on the one hand, or from pharyngeal diphtheria on the other. The frequency of albuminuria, however, is nearly that which belongs to pharyngeal diphtheria, not to non-membranous croup; and another fact which is afforded by one of the cases in this category bears upon the relationship of the laryngeal and the pharyngeal membranous affections. The first-mentioned patient, Jane Suckling, had what in common phrase would be membranous croup, together with follicular spots upon the tonsils. The nurse who tended her after tracheotomy, sitting within the tent and frequently being coughed upon through the wound, became the subject of a

membranous affection of the tonsils, which was too continuous to be called follicular, and to which no name but diphtheria could be given. Hence, it is to be inferred that the child's laryngeal affection was also diphtheritic; and the proof is afforded that in this instance at least membranous croup was simply laryngeal diphtheria.

In class 4, where, together with laryngeal membrane, there was extensive membrane in the pharynx, the association with simply pharyngeal diphtheria becomes more marked. Cold was supposed to have given rise to the attack in only one of ten cases; stench and foul water were each mentioned in one instance, and the relationship to the zymotic diseases was further declared by the record in more than a fourth of premonitory fever or malaise. It is to be noted that among these cases was one in which the disease was probably, and another in which it was certainly, produced as part of an attack of scarlatina.

Class 5 represents cases of undisputed pharyngeal diphtheria, and only concerns the questions now before the Committee as supplying a standard of comparison. Cold takes no place among the hypothetical causes. Nine out of the fifteen cases were traced conclusively, or with merely probability, to foul air or foul water. It is to be observed that in a large proportion of cases the contents of drains, water-closets, cesspools, and dungheaps appear to have been concerned in the production of the disease, whether by means of poisoned water or foul air. Two of the fifteen were apparently due to infection, in one case a fatal attack of pharyngeal diphtheria gave rise to pharyngeal diphtheria in the medical attendant, who had been closely employed in dressing the throat, in the other, a child with laryngeal diphtheria (Suckling, Table 3), gave pharyngeal diphtheria to her nurse. Some of the cases have interest as showing a relationship between the diphtheritic and other morbid influences. The inmates of a farmhouse, including eight children, drank of a well into which drain refuse entered; a man and woman who lived near drank of the same water. The man and woman both had severe and protracted diarrhoea. Of the children one died of erysipelas with some affection of the throat, another (a new born infant) of purulent ophthalmia, together with inflammation about the navel, and four had diphtheria, in three cases confined to the pharynx, in one entering the larynx with a fatal result. The three children who died did so within the space of one week, and the three who recovered were ill at the same time. Whether the several disorders were derived by infection from the person or by means of the water, these cases at least suggest that diarrhoea, erysipelas, purulent ophthalmia, and diphtheria may be produced in different persons, either by the same influence or by influences which are derivable from each other.

Another point of morbid relationship which these tables exem-

plify, though there is not wanting other evidence to the same effect, is that follicular tonsillitis may be derived from diphtheria, as indicated by a continuous membrane, or may have a common origin with it.

The only natural division markedly indicated in this series of cases is into non-membranous and membranous disease. But it is also manifest that the history and general symptoms of the membranous cases differ according to the part attacked, the evidence of contagion and of constitutional affection being generally less marked where the disease is chiefly laryngeal than where it is chiefly pharyngeal.

Some remarks on Dr. Dickinson's tables. By Dr. HILTON FAGGE.

It appears to me important that Dr. Dickinson's tables should be studied from both points of view—(1) that of those who hold that all membranous croup is diphtheria, and (2) that of those who hold that there is a membranous croup distinct from diphtheria, but constituting one disease with (at least) the more severe forms of non-membranous croup.

As I at present hold the second view, I propose to discuss the tables in order to see whether they are not as consistent with it as with the first view.

I may point out that the *form* of the tables is such as to favour rather the first view, by making it more simple of application, since it would place the boundary line between Series I and Series II; whereas, if one holds the second view, it is still a question whether the boundary line lies between Series II and Series III, or within Series III itself, or between Series III and Series IV.

Even on the first view, however, the application of the tables is not quite so simple as at first appears. Dr. Dickinson himself remarks that it is a question whether in the case of Daniel O'Connell (which is included among the non-membranous cases) membrane was not really present. The urine was albuminous, and one might be inclined to think that this really was a case of diphtheria, but for the long duration of the disease (fifteen days) before the child was taken away from the hospital by the friends.

But, in other respects, I cannot agree with Dr. Dickinson that between Series I and Series II, "a natural division is markedly indicated." To me it seems that the facts are just what one should expect, if the division were altogether an artificial one, Series I including those cases of croup too mild to be attended with the formation of membrane. For only one case proved fatal, and of the rest a large number could at once be set down

as being probably "spurious," by their recurring again and again in the same individual, or by their sudden onset.¹

The most striking exception is the case of E. Lacon. Now with regard to this case it is to be noted that the evidence, as to the absence of membrane stands in very nearly the same position as it would have stood in three or four out of the six cases in Series II, if in the latter there had not been a post mortem to show the presence of membrane. There is not a single one of the cases in Series II in which membrane was seen before tracheotomy, or before death, except that of Marrener Hutson (which is proved by several heads of evidence to have been one of laryngeal diphtheria). It may be said that if the cases in Series II had lived long enough, or had recovered, the membranes must have been expelled; but I am not at all convinced of this.

Turning now to the question of the relation between Series II (of membranous laryngitis confined to the air-passages) and Series III—V, it seems to me that Dr. Dickinson's facts are quite consistent with the (second) view that whereas Series V and IV contain none but cases of diphtheria, Series II and Series III, one or both, contain a mixture of cases of laryngeal diphtheria with cases of an independent membranous croup.

¹ Dr. Dickinson has shown that in *some* cases in which membranous exudation exists in the larynx there is a sudden commencement of symptoms in the night; but I think that the presumption is in favour of the attack being more alarming than dangerous when it commences in this way.

APPENDIX VI.

DR. HILTON FAGGE'S COLLECTION OF CASES.

THINKING that a summary of the experience of a large metropolitan hospital would be useful to the Committee, I asked Mr. W. H. Lamb, M.B., who was my clinical assistant in 1877, to collect, from the volumes of medical, surgical, and pathological records, all the cases of croup and diphtheria which he could find. Subsequently I published short notes of them in the 'Guy's Hospital Reports' for 1877, believing that this would render them more available for the objects of the Committee than they otherwise would be.

In the following year Dr. Gee made a similar collection of cases from the Hospital for Sick Children, but in a somewhat different form. I have now placed my cases under the same tabular arrangement which he adopted, so that they can be readily compared.

The first twenty-two of my cases were, in a certain sense, beyond the aim of the investigations of the Committee, being cases of diphtheria, in which there was no evidence that the morbid process extended to the air-passages. Dr. Gee did not include such cases in his tables, and I have therefore now omitted them from mine. But I may state that they were very heterogeneous in their character. Seven of them only were undoubted cases of genuine diphtheria, admitted into the wards for that disease; eight of them arose by contagion in patients already in the hospital for some other complaint; the remaining seven were cases of diphtheria secondary to pyæmia or Bright's disease, or were altogether doubtful in their nature. It will subsequently appear that this small number of cases of indisputable pharyngeal diphtheria probably has a somewhat important bearing on the question of the relation of membranous croup to that disease.

CLASS I.

TABLE I.—*Cases of diphtheria in which the air-passages were involved,*

No.	Date of admission.	Ætiology.	First symptoms of diphtheria.	Membrane seen on fauces.	Laryngeal symptoms.	Tracheotomy.
23	Aug. 13, 1876	Child had been in house where another had died of diphtheria	—	None	Dyspnœa	Yes
24	Aug. 8, 1876	No data	Slight spasmodic cough	Throat could not be seen	From first	Yes, Aug. 9
25	Aug. 30, 1875	Ten days before one sister died	Aug. 16, cold and croupy cough	Soft palate	None on admission, September 2	Yes, Sept. 2
26	Sept. 15, 1873	Attributed to getting wet	On admission	Tonsils and uvula, milky-looking secretion, Sept. 15, shred adherent also to uvula	On admission	Yes, Sept. 16
27	July 16, 1872	Recently had measles and scarlatina	July 14	None	Ditto	Yes, July 16
28	Sept. 3, 1887	One of four children, all died	—	—	No other history	—
29	Mar. 22, 1862	Two children lost with diphtheria	—	On admission, March 22	—	—
30	May 20, 1874	No data	—	May 20	May 20	May 20
31	Nov. 21, 1873	Ditto	Nov. 15, hoarse dyspnœa and cough	Not mentioned	Nov. 15	Nov. 21
32	Dec. 18, 1860	Three other children in same house had died with similar symptoms; scarlatina in neighbourhood	Dec. 8	Ditto	—	No
33	Oct. 19, 1873	No data	Oct. 17, wheezing	Patch on right tonsil, Oct. 19	Oct. 17, wheezing	Oct. 19

SECTION 2.

the fauces being at the same time affected to a marked extent.

Urine.	Result.	Post-mortem examination.		Remarks.
		Resp. fauces.	Resp. larynx.	
Albuminous	Death, Aug. 18	Posterior wall of pharynx and nasal angle = whitish-grey mucus	Lined with false membrane; at vocal cords could just be scraped off, below vocal cords completely detached.	
No note	Death, Aug. 9	Membrane	Membrane.	
Slight traces, Sept. 2	Death, Sept. 4	Ulcerations on uvula and free edge of soft palate	At bifurcation of trachea distinct membrane; tenacious also in bronchial tubes (small).	
Albuminous	Death, Sep. 17	No post mortem.		
Albumen, July 19	Death, July 20	Soft palate, grey lymph thickened	Whole larynx affected; ulcer below left vocal cord	Disease spread to another patient
	Death, Sept. 4	Membrane on soft palate, fauces, pharynx	Thick false membrane.	
Albumen with casts	Death	Tonsils, pharynx, soft palate, a thin membrane	Epiglottis and glottis swollen; on vocal cords and trachea small pieces of membrane.	
No data	Death, May 21	Mucous membrane, fauces, tonsil, uvula, &c., of a yellowish hue	Larynx and trachea full of brownish pus.	
Ditto	Death, Nov. 22	Membrane on right tonsil and lateral and posterior wall of pharynx	Larynx stuffed full of lymph; whole trachea lined with lymph.	
No data; hæmorrhage from bladder	Death, Dec. 20	One tonsil enlarged	Upper part of larynx and vocal cords enlarged; both sides of epiglottis and vocal cords covered with lymph	Hæmorrhage from bowels.
No data	Death, Dec. 23	Patches of lymph on pharynx and right tonsil	Membrane lining whole of larynx and extending into bronchi.	

130 REPORT OF THE COMMITTEE ON THE RELATIONS OF

No.	Date of admission.	Ætiology.	First symptoms of diphtheria.	Membrane seen on fauces.	Laryngeal symptoms.	Tracheotomy.
34	June 2, 1869	No data	May 30	No data	—	June 2
35	Oct. 18, 1872	Ditto	Oct. 16, dyspnœa	Back of fauces	Laryngeal	May 19, 1.30 a.m.
36	Nov. 14, 1872	Ditto	Swallowed with difficulty one month ago	No data	No data	Yes
37	July 21, 1869	Ditto	July 18, dribbling	Ditto	July 21, dyspnœa	July 22
38	July 7, 1876	Ditto	Dyspnœa	Ditto	—	Yes
39	April 14, 1865	Ditto	No data	Ditto	No data	No data
40	Mar. 19, 1864	Ditto	Ditto	Ditto	Ditto	March 19 (on admission)
41	April 14, 1863	Ditto	Sore throat and croup, April 7	Ditto	Ditto	No data
42	Aug. 7, 1861	Ditto	No data	Ditto	Ditto	Ditto
43	Feb. 28, 1872	—	—	—	—	—
44	Nov. 6, 1873	—	—	—	—	—
45	Mar. 21, 1862	Caught in hospital	—	—	—	—
46	Oct. 18, 1874	Ditto	Sore throat just before she was transferred	—	—	—
47	Aug. 10, 1864	Ditto	—	—	—	—

Urine.	Result.	Post-mortem examination.		Remarks.
		Resp. fauces.	Resp. larynx.	
No data	Death, June 2	False membrane from posterior nares to cricoid, then from epiglottis through larynx to bifurcation	On fauces, stuck tightly	Brought in, in a dying state.
Ditto	Recovery, Nov. 20	—	—	Each time canula was drawn out lymph, which left a cast.
No albumen	Death, Dec. 17	Patches of lymph on tonsils	Larynx swollen; patches of lymph.	
No data	Death, July 24	No report of post-mortem.		
Ditto	Death	False membrane, soft palate, tonsils	False membrane, larynx, trachea, bronchi could be drawn out easily in casts from bronchi and divisions	Kidneys congested, excess of epithelium.
Ditto	No data	Soft palate, tonsils, pharynx, covered with false membrane (patches)	Epiglottis, larynx, trachea covered with a firmly adherent continuous layer of lymph	Lobular pneumonia found at post mortem.
Ditto	Death, Mar. 20	Lower part of tonsils, membrane continuous to epiglottis	Membrane on both aspects of epiglottis and in larynx and trachea	A little lobular pneumonia at post mortem.
Ditto	Death, Apr. 15	Tonsils, palate, false membrane firmly adherent	Epiglottis, larynx, vocal cords, trachea, a layer of false membrane easily separable	Purulent fluid in bronchi; no tubercle.
Ditto	Death, Aug. 8	Tonsils, false membrane as thick as a shilling, foetid ulcers	False membrane, larynx, trachea, bronchi, lost in purulent pus.	
—	Death, Mar. 1	Diphtheritic	Edematous diphtheritic membrane.	
—	Death, Jan. 26	Thick membrane on epiglottis; extended from pharynx, through larynx and trachea, into smaller bronchi.		
—	Death, Apr. 10	Palate, pharynx, exterior of glottis, covered with false membrane; membrane extended as far as vocal cords; trachea, &c., healthy		Double psoas abscess; diseased spine.
Albumen, sp. gr. 1022	Death, Oct. 23, exhausted	Epiglottis, vocal cords, soft palate, tonsils large; no disease or membrane		In Dorcas Ward some months previously for talipes equinus
Albuminous, highly so	Death, Nov. 26	Palate, tonsils, pharynx, to œsophagus, covered with false membrane; membrane continued below vocal cords into trachea and bronchi; large abscess in left thigh; commencing waxy change		Compound fracture of leg.

TABLE II.—*Cases in which the air-passages were mainly attacked,*

No.	Date of admission.	Ætiology.	First symptoms of diphtheria.	Membrane seen on fauces.	Laryngeal symptoms.	Tracheotomy.
48	Sept. 1, 1876	No data	—	—	Aug. 31	Sept. 1
49	Jan. 9, 1875	—	Cold and violent cough, Jan. 6	None	—	Jan. 9
50	Mar. 5, 1875	—	Cold and hoarseness, Feb. 20	—	—	March 6
51	Apr. 22, 1871	—	April 15, fever and sore throat	—	—	April 23
52	Sept. 9, 1874	—	Sept. 8, croupy cough	Sept. 10, left tonsil	From first	Sept. 10, tube removed on 20th
53	Oct. 18, 1872	Oct. 11, got wet and caught cold	—	—	From first, or nearly so	Oct. 18
54	Sept. 18, 1872	—	—	—	—	Sept. 19, died during operation
55	July 10, 1871	—	Ailing since July 3	—	July 8	July 10
56	Oct. 21, 1867	—	—	—	—	Oct. 21
57	Aug. 28, 1874	—	Aug. 23, cough	—	From first	Aug. 28

the fauces being affected in a very slight degree only, if at all.

Urine.	Result.	Post-mortem examination.		Remarks.
		Resp. fauces.	Resp. larynx.	
—	Death, Sept. 1	Membrane on larynx; membranes with superficial ulceration on labia minora.		
—	Death, Jan. 11	Epiglottis was covered on both its surfaces with a soft layer of yellowish membrane; isolated patches of the same on tonsils and interior of larynx to tracheotomy wound.		
—	Death, Mar. 10	No membrane could be found in any part save trachea		On removing tracheotomy tube membrane was expectorated once or twice.
—	Death, Apr. 23	A few patches of lymph on laryngeal surface of epiglottis.		
Albumen, Sept. 12	Cured, Oct. 31	—	—	
—	Death, Oct. 20	Tonsils swollen, patches of lymph about them	False membrane in larynx adherent to vocal cord.	
—	Death, Sep. 19	Slight membrane over posterior wall of pharynx; tonsils rather worm eaten	Laryngeal aspect of epiglottis covered with white membrane; this extended into larynx and trachea.	
—	—	Small patches of membrane on tonsils	Tough false membrane from larynx to bronchi.	
—	Death, Oct. 23	Tonsils false membrane	Larynx, trachea, bronchial tube, false membrane	Several pieces of membrane came away with tracheotomy tube.
—	Death	—	Upper part of larynx filled with a detached mass of membrane extending to vocal cords	Note by Dr. Taylor.—The tonsils when I saw them appeared to be old enlarged tonsils, slightly inflamed.

CLASS 2.—TABLE III.—*Cases of Membranous Laryngitis of doubtful origin secondary to any other disease*

No.	Date of admission.	Ætiology.	First symptoms of diphtheria.	Membrane seen on fauces.	Laryngeal symptoms.	Tracheotomy.
58	Oct. 28, 1875	From getting wet 14 days ago	Cough	None	From Oct. 27	Oct. 28
59	June 9, 1874	June 5, cold water spilled over him	June 7, cough "barking"	Ditto	From almost the first	June 10
60	Nov. 8, 1873	Nov. 7, slept in a damp room	Nov. 8, 5.30 a.m., cough, breathing wheezing and loud	Ditto	From first	Nov. 8, 10.30 p.m.
61	Jan. 9, 1873	No data	Jan. 8, catarrh, difficulty of breathing	—	Ditto	Jan. 9
62	Mar. 8, 1872	Ditto	Mar. 8, on admission	—	From first, on admission	March 8
63	July 24, 1868	Ditto	Was in a state of extreme dyspnoea on admission	—	Ditto	July 24; in a dying state

as diphtheria, but not directly caused by local injury to the throat, nor of the larynx or trachea.

Urine.	Result.	Post-mortem examination.		Remarks.
		Resp. fauces.	Resp. larynx.	
—	Death, Oct. 29	No membrane in pharynx	Bronchial tubes contained pus; mucous membrane over arytenoids and over pharyngeal aspect of cricoid cartilage injected; trachea very small, narrowed by thickened mucous membrane, could not have admitted a pencil.	
—	Death, June 12	Pharynx healthy; no lymph	From rima to 2 inches below thyroid a delicate, easily detached membrane; beyond this the tubes were full of pus.	
No albumen	Death, Nov. 14	—	A thin layer of lymph below vocal cords as far as bifurcation of trachea	Effect of operation was good, respiration became easy, lividity disappeared, child was soon asleep.
Ditto	Death, Jan. 9, afternoon	Pharynx healthy	Epiglottis on both sides covered with closely adherent membrane; in larynx above vocal cords similar patches	Jan. 10. — In the afternoon child coughed up a plug of membrane.
No data	Death	—	Larynx lined as high as upper edge of epiglottis with distinct membrane; below wound this passed into a more purulent-looking stuff; it extended as low as 2nd division of bronchi.	
Ditto	Ditto	—	Epiglottis, larynx, swollen, the latter having a thick fibrinous cast; false membrane on trachea, hinder surface.	

136 REPORT OF THE COMMITTEE ON THE RELATIONS OF

No.	Date of admission.	Ætiology.	First symptoms of diphtheria.	Membrane seen on fauces.	Laryngeal symptoms.	Tracheotomy.
64	June 15, 1868	No data	Cough for 9 days	—	Just before admission	June 15
65	Oct. 7, 1868	Ditto	Fourteen days before admission; cold, with slight cough	None	n admission	Oct. 7
66	Jan. 9, 1868	Ditto	For week or two had slight cough, wheezing	No data	Ditto	Jan. 9, laryngo-tracheotomy
67	Sept. 22, 1867	—	—	—	—	—
68	Mar. 19, 1865	—	—	—	—	March 19
69	July 31, 1864	—	—	—	—	Died same day on operation table
70	Aug. 13, 1876	—	—	—	—	—
71	Jan. 4, 1856	—	—	—	—	—
72	May 5, 1864	—	—	—	—	May 5, on admission
73	June 16, 1857	—	—	—	—	June 16, on admission

Urine.	Result.	Post-mortem examination.		Remarks.
		Resp. fauces.	Resp. larynx.	
No data	Death	—	Epiglottis and larynx thickened and swollen, posterior surface of former covered with false membrane; tracheotomy tube had pushed false membrane before it, not piercing it, and forming a valve.	
Ditto	Death, Oct. 9, from asphyxia	—	All the tubes contained prolongation of false membrane.	
Ditto	Death, Jan. 11	—	Upper half of trachea, false membrane thick and tough around vocal cord, reached $1\frac{1}{2}$ in. above glottis.	
—	Death	—	False membrane extending from larynx into bronchi.	
No data	Death, Mar. 21	—	Plastic lymph in larynx and trachea, easily detached and coming off in shreds.	
—	Death, July 31	—	False membrane commenced just above artificial openings, and extended near to bifurcation.	
—	Death as soon as admitted, Aug. 18	—	Slight oedema; a little lymph, a definite broad patch, just below vocal cords.	
—	Death, Jan. 5	—	Whole surface of trachea from superior vocal cords to bifurcation lined by a tenacious white membrane.	
—	—	—	Adherent false membrane in larynx, trachea, and bronchi; whole of air-passages acutely inflamed.	
—	—	—	Under surface of epiglottis, larynx, trachea, and bronchi, covered with false membranes, which adhered in shreds.	

No.	Date of admission.	Etiology.	First symptoms.	Membrane seen on fauces.	Laryngeal symptoms.	Tracheotomy.
74	Jan. 19, 1858	Five weeks ago scarlatina in house; 6 days ago caught cold	Cold, cough, dyspnoea, Jan. 13	—	On admission	Jan. 20
75	Mar. 7, 1871	—	Cough, cold, dyspnoea, admission	—	—	March 8, child became easier
76	Dec. 10, 1869	Cough for 1 month	—	—	—	—

CLASS 8.—TABLE IV.—Cases of Laryngitis having a clinical resemblance

77	Jan. 28, 1868	Cold last week	Suddenly taken ill at 2 a.m., Jan. 28	—	—	Jan. 28, brought the child round
78	Oct. 6, 1869	Three weeks ago had scarlatina	Oct. 29, cold and dyspnoea	—	Jan. 29	Jan. 29, at noon, during operation child vomited freely, appeared much relieved
79	Mar. 9, 1867	No data	Cold for some days	None	On admission, Mar. 9	March 9
80	July 12, 1867	—	Cold for some days past	No membrane	On July 12	July 13
81	Mar. 17, 1867	Convalescent of measles, breathing worse since	March 11, slight sore throat	None	—	No
82	Feb. 23, 1869	—	—	—	—	—
83	Apr. 16, 1874	Cough since April 4; no history of infection	—	—	—	—
84	Mar. 24, 1874	Always had cough	Croupy cough and dyspnoea 4 days before admission	—	—	—

MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX VI. 189

Urine.	Result.	Post-mortem examination.		Remarks.
		Resp. fauces.	Resp. larynx.	
—	Death, Jan. 23. suddenly	—	Inflammation of the whole of air-passages	Child's sister, æt. 8, admitted with croup Dec. 12, 1857; tracheotomy performed, and she was sent out Dec. 17.
—	Death, Jan. 11	—	False membrane in larynx, trachea, and bronchi.	
—	Death, Dec. 15	—	Acute plastic laryngitis; membrane adherent to vocal cord.	

to those of croup, but in which no false membrane was found to exist.

—	Death, Jan. 30, from convul- sions	No post mortem.		
—	Oct. 7, death	Ditto.		
No data	Recovery, April 20.			
No albumen	Recovered, Aug. 7.			
No data	Recovered, April 7	—	—	Improved daily.
—	Taken out by parents some- what relieved			
—	Gradually im- proved, and went out May 1.			
—	Gradually im- proved, and went out on April 14.			

140 REPORT OF THE COMMITTEE ON THE RELATIONS OF

No.	Date of admission.	Ætiology.	First symptoms of diphtheria.	Membrane seen on fauces.	Laryngeal symptoms.	Tracheotomy.
85	Oct. 23, 1874	Nineteen days ago first lost her voice	Nineteen days ago dyspnœa; has catarrh	—	—	Oct. 23, 2 p.m. (death seemed imminent just before operation)
86	Jan. 3, 1874	—	Christmas day, wheezing and drowsiness	No membrane	On admission	Jan. 3, dyspnœa so urgent, operation performed (just before operation temp. 97·8°, pulse 168, resp. 36; even. temp. 101·0°, pulse 160, resp. 52)
87	Aug. 26, 1876	—	Two days before admission croupy voice	Ditto	Ditto	Aug. 27, as there was great dyspnœa tracheotomy was performed; marked relief followed
88	Feb. 21, 1876	Child and two others just recovered from morbilli	—	Fauces and soft palate little injected no deposit	On admission paroxysms of ringing cough	No

MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX VI. 141

Urine.	Result.	Post-mortem examination.		Remarks.
		Resp. fauces.	Resp. larynx.	
Albumen, 25th, urine normal	Discharged well, Nov. 24.			
No albumen	Went out well March 27	—	—	Was in hospital till Feb. 27; readmitted on Feb. 28, owing to râles heard in chest.
No data	Discharged, Sept. 25	—	—	Continued to improve daily after operation; tube removed Sept. 5.
Ditto	The brassy cough gradually diminished in frequency. Mar. 1st, went out	—	—	

The paper in the 'Guy's Hospital Reports' contained a fourth class of cases, which, like the first twenty-two cases, correspond with more of Dr. Gee's cases; namely, cases of membranous laryngitis, with or without pharyngitis, directly caused by local injury to the throat, or secondary to pre-existing local disease. Of cases due to injury there were eleven; five were secondary to some local disease of the air passages or lungs. For details of, refer to the 'Reports.'

The following are the conclusions which I thought I might legitimately draw from my cases, without prejudice to the main question before the Committee.

"In the first place, I think that the cases in Class IV negative the *à priori* argument that the mucous membrane of the air-passages is not likely under simple (or non-specific) irritation to take on an inflammatory process attended with the formation of false membranes. There are recorded sixteen instances in which a membranous laryngitis was developed as the result of scalds by hot water, after the entrance of a foreign body into the trachea, after a cut throat, after tracheotomy for various conditions, or secondarily to some disease of the air-passages. Some of the cases in question might, indeed, be plausibly attributed to infection from tracheotomy instruments, if we were to suppose those instruments to have been previously employed for cases of diphtheria, and to have been insufficiently cleaned, Mr. Howse has told me that he believes this to have occurred in Case 89, which came under his observation at the time. But this explanation goes only a very little way.

"Secondly, the cases in Class IV show that, great as is the anatomical difference in structure between the pharyngeal mucous membrane and that which lines the larynx, it is no barrier to the transference of morbid action from the former to the latter surface. The same thing is notoriously true as regards diphtheria. But if a plastic laryngitis may be set up by extension downwards from the pharynx, one does not see why a membranous pharyngitis may not be consecutive to a similar affection of the air-passages; in other words, there is no reason for supposing that a simple membranous croup (if such an affection exists) may not be attended with the formation of false membrane upon the fauces.

"Thirdly, the cases of laryngitis recorded in Class III, in which no false membranes were proved to have been formed, do not appear to have differed notably in their clinical features from those in Class II, in which false membranes were discovered after death, or expectorated during life. So far as the imperfection of these Reports enables us to judge, there is only one case (Case 77) which presented any of the characteristic of 'stridulous laryngitis' in 'spurious croup.' It seems to me that this affection as it is described by French writers has a fair claim

to be considered a separate member of the nosology, its distinguishing features being its liability to return again and again in the same patient, and the suddenness of its commencement, with symptoms which from the very first are of the most alarming character, but which quickly subside and never lead on to a persistent attack, lasting for several days without intermission. Unless, however, we insist upon these characteristics, I can perceive no valid reason for drawing a boundary line between those cases of croup in which membranes are, and those in which they are not, found to exist. In many of the former cases they are not discovered until a post-mortem examination is made. Now, the latter cases are seldom, if ever, fatal. I cannot find in our records a single case of croup in which the patient died and no false membranes were found.¹ If, on other grounds, membranous croup can be shown to be always a laryngeal diphtheria, the distinction between the two sets of cases is of course necessary. But unless this can be done, to suppose that such a distinction exists is almost the same thing as to assume that a disease, when it is fatal, is attended with morbid changes essentially different from those which characterise it when recovery takes place. And I submit that this is altogether without precedent in pathology. Considering that in every instance in which false membranes are found in the air-passages, they shade off into muco-purulent matter in the trachea or bronchi,—and that, in some instances, there are only small shreds of lymph imbedded in such secretion within the larynx itself,—it is surely very improbable that the presence or absence of false membranes forms an absolute distinction between two entirely different diseases. My own opinion is that the cases in Class II and those in Class III should be associated together under the common name of croup, assuming always that it is not proved that those in Class II belong to diphtheria.

“Fourthly, we now come to the question as to the relation between the cases in Class II and those in Class I; and at first sight there seems to be a very marked contrast between them. In Class I we have a highly infectious disease, of which albuminuria is a very frequent symptom, and which is often attended with swelling of the cervical glands. In Class II we have a disease which seems not in a single instance to have arisen in contagion, nor to have spread to other patients; in only one of the nineteen cases of this class was albuminuria noticed to have been present; and I think there is only one in which the glands are said to have been swollen.

¹ Since this was in type I have made a post-mortem examination in a case of croup, in which death occurred after fifty-one hours' illness, and in which the larynx was perfectly healthy, but the trachea and bronchi contained a soft, viscid, muco-purulent material, without even any shreds of false membrane in it.

"But I am bound to say that a closer analysis of the cases in Class I throws some doubt upon the validity of the distinctions to which I have just referred; and I must acknowledge my indebtedness to Dr. Greenfield for having suggested to me certain objections which might fairly be made to them.

"The first point is the preponderance of children among those cases in Class I, in which diphtheria spread to the air-passages. Among the cases contained in Section 2 of Class I there are twenty patients who came into the hospital suffering from diphtheria; five who caught it while in the wards. The latter were adults; but of the former twelve were under the age of five years, four between the ages of five and fifteen, and four above fifteen years old. And all but one of the cases in Section 3 were those of children under five years. Again, there has not been a single case in which a child below that age has died of diphtheria in the hospital, and has been found to have its larynx free on post-mortem examination. This excessive liability of children to be affected with the laryngeal form of diphtheria, at the very age which has been generally supposed to be that at which croup is most apt to occur, may certainly be made a point in favour of the identity of the two diseases.

"Again, we find a difference in the extent to which the cases in Sections 1 and 2 respectively can be brought into connection with other cases of diphtheria, either as having caught the disease from them or given it to them. Among the fifteen indisputable cases of diphtheria in the former section there are eleven in which such a connection can be traced. But of the twenty-five cases in the latter section there are only eight in which the existence of a contagious or epidemic influence is recorded. One therefore is not surprised to find that not one of the cases in Section 3 afforded an instance of the manifestation of such an influence. It undoubtedly seems as though diphtheria were less contagious in proportion as the fauces are less severely affected. The comparatively rapid course and early fatal termination of the cases in which the disease extends to the air-passages suggests itself as an explanation, but I doubt whether it is a satisfactory one. The only way of interpreting the cases in Class I by which one could avoid this conclusion would be by supposing that a non-specific membranous croup may be attended with the formation of extensive patches of false membrane on the fauces; in other words, that a large number of the cases in Section 2, and almost all those in Section 3, are not instances of diphtheria at all. This is a question to which I shall presently return.

"But, if it be true that laryngeal diphtheria is comparatively little contagious, one can hardly attach much importance to the fact, which at first sight appeared so striking, that no contagion can be traced in any of the cases of membranous laryngitis in Class II, which I have classified as of doubtful origin.

"Another point on which I was at first inclined to lay great stress is that no instance of membranous laryngitis, apart from pharyngeal diphtheria, has occurred among those cases in which the disease has arisen by contagion in persons already in the hospital. But it may be argued that if laryngeal diphtheria is peculiar to children, one would hardly expect to find it developing itself in the wards of a hospital like Guy's, in which the great majority of the patients are adults. Still there are some children in almost every division; and I think that some importance may fairly be attached to the fact just stated. The experience of a hospital specially devoted to children's diseases would be of great value.

"Other points of distinction failing us, we can fall back upon the general numerical ratio between the cases of recognised diphtheria and those of membranous laryngitis admitted into the hospital, and we may ask whether there are not too many of the latter to be set down as instances of an exceptional variety of the former disease. Now, so far as I know, the only trustworthy statements as to the frequency with which diphtheria when epidemic attacks the larynx without at the same time affecting the pharynx or tonsils are those of Bretonneau and Guersant.¹ Guersant ('Syd. Soc. Memoirs,' p. 216) says that the number of such cases may perhaps amount to a twentieth of all cases of diphtheria, but he implies that unless the fauces are inspected from the very commencement of the disease the presence of slight membranes upon them is apt to be overlooked. Bretonneau relates only one case of what he believed to be purely laryngeal diphtheria among the forty-five recorded in his papers on the subject; and in that instance (p. 165 of Syd. Soc. translation) there is really no proof that the disease was diphtheria rather than simple croup. He goes on to say that it was the second time, and in the proportion of one to thirty, that he had met, after death, with diphtheritic inflammation limited to the air-passages.

"At Guy's Hospital we seem to have had nineteen cases of membranous laryngitis to fifty-seven of diphtheria. It is true that the reports of many of the former cases are imperfect; but I do not think it is likely that the clinical clerks have often failed to note down the presence of false membranes upon the fauces, where any have been detected; and as their absence has for years past been regarded as the crucial distinction between the two diseases, they are certain to have been looked for. But

¹ Since this was written I have read Dr. Yeats' account of an epidemic which occurred at Auchtergaven in Perthshire ('Ed. Med. Journ.,' 1876). Among 183 cases there were 15 in which laryngeal symptoms were present from the commencement, but in which there was no visible affection of the fauces, when they were first brought under notice; and in 6 of these the pharynx remained free during the whole progress of the disease.

whatever deduction should be made, on the score of incompleteness, from the cases of membranous laryngitis, a large deduction must also be made from those of diphtheria before a fair comparison can be instituted. For in ten of the latter cases this disease arose by contagion in persons already in this hospital; and Class I includes several other cases of which the real nature is altogether doubtful. Now, I do not see any reason why diphtheria should attack the larynx more often when it is sporadic than when it is epidemic. I, therefore, must regard the relatively large number of cases of membranous laryngitis as a weighty argument in favour of the separate existence of a membranous croup.

"Hitherto I have argued the question on the basis that the presence of patches of false membranes on the fauces proves a case to be one of diphtheria. But, after all, this is an assumption, and one which, as I have already shown, is rendered improbable by the fact that in the cases in Class IV the pharynx and the larynx have frequently been found to be simultaneously affected. Between the years 1839 and 1849, long before epidemic diphtheria was prevalent in London, Dr. West found that the velum and tonsils presented false membranes in a considerable proportion of his cases of croup. I am not at all sure that the real solution of the difficulty may not be found in abstracting from diphtheria a considerable number of the cases in Section 3, and even some of those in Section 2, of Class I. We should then get rid of the puzzling anomaly that the disease seems to be so much less contagious when it mainly affects the larynx than in the ordinary pharyngeal variety.

It is possible that a further head of evidence in regard to the question of the relation of membranous laryngitis to diphtheria may be found in the proportionate number of males and females especially attacked by these diseases. All writers say that croup is more common in boys than in girls. This is confirmed by the cases in Class III, as regards the affection in which no false membranes are developed. But diphtheria is equally prevalent in the two sexes. If, therefore, males should preponderate among those who suffer from membranous laryngitis, one would be disposed to associate it with croup; if not, one would rather take it for a form of diphtheria. Now, in Class II there is no excess of boys; but it is curious that they do preponderate among the cases in Section 3 of Class I.

"Let me recapitulate, in somewhat different language, the main conclusions to which the facts recorded in this paper appear to lead us:—We find that the attempt to separate from diphtheria a membranous croup in which the fauces remain entirely free from false membranes is beset with difficulties. The cases (which must then be called cases of diphtheria) in which the air-passages are attacked, the palate and tonsils being

but slightly affected, occur almost exclusively in children; and they are seldom, if ever, infectious, whereas pharyngeal diphtheria is highly infectious. But when one has once admitted that the different forms of diphtheria present different degrees of infectiousness, and that each of them occurs with special frequency at a particular period of life, one is debarred from insisting on the sporadic character of membranous laryngitis, and the fact that it never arises in the wards of a general hospital, as proof that it is distinct. It is otherwise if we draw the boundary line, not between the cases in Class I and those in Class II, but within Section 2 of Class I itself; allowing that the non-specific, simply inflammatory affection may be attended with the formation of false membranes even on the fauces. Such a view does away with the very improbable supposition that laryngeal diphtheria differs from the ordinary form of the disease in being peculiar to children, and in possessing little or no infectiousness; and I think that it commends itself to us on other grounds also."

DR. FAGGE'S CASES.

CLASS I.—*False membrane in larynx and trachea.*

Sex.—Males, 15. Females, 20=35.
 Age.—1-3 years. 2-3. 3-4. 4-5. 5-6. 7. 10+. 16. 18. 24. 26.
 6 7 3 7 2 1 1 1 1 1 1
 34. 41. 42.
 2 1 1=35.

Time of year (month of admission):

Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.
1	1	4	3	1	1	4	6	5	5
Nov. Dec.									
3 1.									

Etiology:

Other cases of diphtheria in neighbourhood, 5.
 Contracted in hospital, 3; gave the disease to other patients, 2.
 Catching cold, 1; measles + scarlatina, 1.
 No data, 23.

Earliest local symptoms:

Sore throat, 2.
 Croupy cough, hoarseness, dyspnoea, 10.
 Sore throat + laryngeal symptoms together, 1.
 Coryza and febrile symptoms, 3.
 No data, 19.

Time of onset of laryngeal symptoms.

Tracheotomy, 24.

Albuminuria:

Present, 8.

Absent, as often as examined, 1.

No data, 26.

Result:

Died, 32.

Recovered, 2.

Not known, 1.

CLASS II.—*False membrane in air-passages alone.*

Sex.—Males, 10. Females, 8. Not stated, 1=19.
 Age.—1-3. 2-3. 3-4. 4-5. 5-6. 6-7.
 7 6 2 1 2 0 Not stated, 1=19.

Time of year:

Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.
4	0	3	0	1	3	2	1	1	2
Nov. Dec.									
1 1=19.									

Etiology:

Exposure to cold, 4.
 Contracted in hospital, 0.
 Diphtheria in neighbourhood, 0.
 No data, 15.

Earliest local symptoms:

Sore throat.
 Laryngeal, 6.

Coryza, 2.

No data, 10.

Time of onset of laryngeal symptoms.

Tracheotomy, 15.

Albuminuria:

Present, 0.

Not present, 2.

No data, 17.

Result:

All fatal.

APPENDIX VII.

DR. GEE'S TABLES.

CLASS 1.—*Cases of membranous laryngitis associated with a similar condition of the fauces. Hospital for Sick Children from 1853 onwards.*

Dr. Buchanan's Case-book.

23.	.	Hannah Lovibond.	2 years.	Death.	Alb.
24.	.	Alice Johnson.	1½ "	" "	No alb.

Dr. Hillier's Case-books.

25.	Vol. i, p. 481.	"Dr. Babington's case."	"	p.m.	No alb.
26.	328.	Florence Keyte.	2 years.	" "	?
27.	446.	Thomas Ryan.	2 "	" "	?
28.	Vol. iij, p. 55.	Edward Gibbons.	3 "	" "	?
29.	79.	Alfred Bransgrove.	4 "	" "	?
30.	59.	Mary Griffin.	4½ "	Recovered.	Alb.
(Membrane expectorated.)					
31.	212.	M. Little.	2 years.	Death. p.m.	?
32.	74.	John Lane.	3 "	" "	Alb.
33.	Vol. iv, p. 152.	William Smeaton.	2 "	" "	"
34.	176.	Frances Shiel.	8 "	" "	"
35.	170.	Lydia Grimbley.	4 "	" "	"
36.	186.	Ellen Groom.	3½ "	" "	"
37.	158.	Frederick Vihw.	2½ "	" "	"
38.	Vol. v, p. 173.	Joseph Wing.	5 "	" "	"
39.	167.	Mary Stanning.	3½ "	" "	"
40.	Vol. vi, p. 44.	Anne Gee.	1½ "	" "	?
41.	Vol. vii, p. 184.	Theresa Durham.	2½ "	" "	Alb.
42.	218.	Louisa Bird.	3 "	" "	"

Dr. West's Case-books.

1.	Vol. i, p. 59.	F. Hackney.	6 "	" "	?
2.	469.	James Walton.	3 "	" "	?
3.	325.	Thomas Vose.	3 "	" "	?
4.	71.	Bridget Buckley.	5 "	" No p.m.	?
(Membrane expectorated.)					
5.	241.	Emily Carpenter.	5 years.	Death. p.m.	?
6.	Vol. ii, p. 343.	John Jones.	2½ "	" "	?

MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX VII. 151

7.	Vol. iii, p. 23.	Elizabeth Duckett.	2 years.	Death.	p.m.	?
8.	357.	Flora Cogswell.	2 $\frac{1}{2}$	"	"	Alb.
9.	Vol. v, p. 91.	Emily Walker.	4 $\frac{1}{2}$	"	"	"
10.	115.	Clara Cook.	5	"	"	"
11.	Vol. vi, p. 81.	Maria Cracknell.	2	"	"	"
12.	241.	Frederick Freeth.	3 $\frac{1}{2}$	"	"	No alb.
13.	319.	Charles Winton.	5	"	"	?
14.	15.	Henrietta Esterby.	3	"	"	Alb.
15.	73.	John Harker.	6	"	"	"
16.	Vol. vii, p. 37.	Henry Norton.	2 $\frac{1}{2}$	"	"	?
17.	361.	Florence Ralph.	4 $\frac{1}{2}$	"	"	Alb.
18.	1.	Florence Stowe.	1 $\frac{5}{8}$	"	"	"
19.	Vol. ix, p. 171.	Janet Murphy.	2 $\frac{1}{2}$	"	"	?
20.	411.	John Hope.	2	"	"	?
21.	345.	Elizabeth Wright.	3 $\frac{1}{2}$	"	"	?
22.	Vol. x, p. 135.	Charles Conroy.	13 months.	"	"	?

CLASS 2.—*Cases of membranous laryngitis not associated with a similar condition of the fauces.*

Dr. Hillier's Case-books.

1.	Vol. iv, p. 156.	M. Huggins.	5 years.	Death.	p.m.	Alb.
2.	Vol. v, p. 182.	George Miller.	1 $\frac{1}{2}$	"	"	?
3.	184.	Gertrude Fachler.	6	"	Recovery.	
(Membrane expectorated.)						
4.	Vol. vi, p. 31.	Elizabeth Bass.	5 years.	Death.	p.m.	?
5.	48.	Elizth. McCarthy.	4	"	No p.m.	Alb.
(Membrane expectorated.)						

Dr. West's Case-books.

	Vol. iii, p. 41.	William Gray.	2 $\frac{3}{4}$ years.	Death.	p.m.	?
6.	Vol. i, p. 51.	J. G. Cassaigne.	11 $\frac{1}{2}$	"	"	?
7.	543.	Edward Dolman.	3	"	"	?
8.	559.	Henry Hoare.	5	"	"	Alb.
9.	Vol. ii, p. 29.	Emma Manley.	1 $\frac{1}{8}$	"	"	?
10.	Vol. iii, p. 385.	John Knight.	2 $\frac{1}{2}$	"	"	?
11.	445.	Mary Stacey.	7	"	"	No alb.
12.	109.	Charlotte Holmes.	3	"	"	?
13.	Vol. iv, p. 471.	Caroline Huggard.	5 $\frac{1}{2}$	"	"	Alb.
14.	241.	George Lepine.	2 $\frac{3}{4}$	"	"	"
15.	Vol. vi, p. 165.	Fanny Smith.	4	"	Recovered.	"
(False membrane expectorated.)						
16.	545.	Fred. Norman.	5 years.	Death.	p.m.	"
17.	Vol. vii, p. 147.	Mary Connor.	4	"	"	?

18. Vol. viii, p. 95. Alfred Bunn.	3½ years.	Death.	p.m.	No alb.
19. 141. Hattie Fairbank.	4 "	"	"	?
20. Vol. ix, p. 209. Fred. Allwright.	3½ "	"	"	?
21. Vol. xi, p. 163. Thomas Brown.	2½ "	"	"	?

(Doubtful case.)

CLASS 3.—Cases of acute laryngitis, whether catarrhal or membranous unknown.

Dr. Hillier's Case-books.

Vol. i, p. 336. Henry Sylvester.	9 years.	Recovered.	Alb.
		(Membrane on fauces.)	
414. William Wilcox.	2½ years.	Recovered.	
		(Membrane on fauces.)	
497. Timothy Collinson.	4 years.	Recovered.	
		(No membrane seen.)	
Vol. ii, p. 212. James Martin.	4 years.	Recovered.	
		(Membrane on fauces.)	
Vol. iii, p. 78. Mary Lane.	1½ years.	Died.	p.m.?
		(Membrane on fauces.)	
84. Alice Randal.	19 months.	Recovered.	
		(No membrane seen.)	
Vol. v, p. 190. John Wiggins.	5½ years.	Recovered.	
		(No membrane seen.)	
Vol. vii, p. 34. Benjamin Thomas.	5½ years.	Recovered.	
		(No membrane seen.)	

Dr. West's Case-books.

Vol. i, p. 91. Richard Smith.	1½ years.	Recovered.	
		(No membrane seen.)	
163. Edwin Grant.	5 years.	Recovered.	
		(Membrane on fauces.)	
417. George Coadar.	4 years.	Recovered.	
		(Membrane on fauces.)	
Vol. ii, p. 309. Eliza Brough.	2½ years.	Recovered.	
		(Membrane on fauces.)	
445. Matilda Pike.	1½ years.	Recovered.	
		(No membrane seen.)	
357. Arthur Fortune.	2½ years.	Recovered.	
		(Membrane on fauces.)	
Vol. iv, p. 493. Walter Dunkin.	5 years.	Recovered.	
		(No membrane seen.)	

MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX VII. 153

Vol. iv, p. 97.	Thomas Antony.	2 years.	Died. p.m. (Membrane on fauces, died late on.)
Vol. v, p. 127.	Mary Lucas.	3 years.	Died. No p.m. (Membrane on fauces.)
Vol. vi, p. 161.	Edward Wells.	3 years.	Recovered. (No membrane seen.)
Vol. vii, p. 321.	Sarah Saunders.	4 years.	Recovered. (No membrane seen.)
Vol. viii, p. 153.	William Timpin.	2 $\frac{1}{2}$ years.	Recovered. (No membrane seen.)
	367. Kate Peters.	5 $\frac{1}{2}$ years.	Recovered. (No membrane seen.)
Vol. ix, p. 375.	Sarah Blake.	3 $\frac{1}{2}$ years.	Recovered. (No membrane seen.)
	415. John Hines.	5 years.	Recovered. (Membrane on fauces.)
Vol. x, p. 569.	Jos. Blackberough.	7 years.	Recovered. (No membrane seen.)

CLASS 1.—*Cases of membranous laryngitis,*

No.	Date of admission.	Ætiology.	First symptom of diphtheria.	Membrane seen on fauces.	Laryngeal symptoms.
1	Sept. 28, 1858	Father and brother had same disease	Sore throat, Sep. 26	Sept. 28 (admission)	Oct. 4
2	June 21, 1861	No data	Croupy cough, June 18	Not during life	From first
3	MAR. 26, 1861	Ditto	Ill, Mar. 17; sore throat, Mar. 20	Tonsils, pharynx; on admission	Mar. 24
4	Mar. 1, 1859	Ditto	Ill, Feb. 27; dyspnoea, Feb. 28	Tonsils; "slight whitish deposit"	Feb. 28
5	Jan. 20, 1861	Contracted in hospital. Abscess in thigh; rheumatism? pleurisy and pericarditis; acute nephritis (Jan. 22); erysipelas of thigh, Jan. 24	Croupy cough, Jan. 31	No note	Jan. 21
6	Oct. 18, 1861	Contracted in hospital. Tubercular meningitis; "diphtheria prevalent in ward"	Sore throat, Oct. 27	Ditto	None
7	Feb. 6, 1862	Contracted in hospital. Polyuria; scarlet fever, Feb. 23	Swallowing and breathing difficult Mar. 17	None seen	From first
8	Dec. 8, 1862	Sister had diphtheria afterwards	Sore throat, Dec. 3	Child not seen during life	Dyspnoea, Dec. 7
9	July 30, 1863	Contracted in hospital. Febricula, July 28	Sore throat, Aug. 8	Tonsils, Aug. 8	Croupy breath 8 hours after sore throat
10	Aug. 11, 1863	A child living in same house had diphtheria	"Slight cold," Aug. 6; sore throat, Aug. 7	Tonsils, pharynx; on admission	Croupy breathing, Aug. 10
11	Apr. 16, 1864	Attributed by mother to going out insufficiently clothed	"Cold on chest," Apr. 18; croupy cough, Apr. 15	A few white specks on tonsils; no false membrane until Apr. 17	Apr. 15
12	Sept. 5, 1864	Nothing discoverable	Febrile, Aug. 31; croupy breathing, Sept. 3	Sore throat, Sept. 4; no false membrane on admission	Sept. 3
13	Nov. 19, 1864	Cough 2 weeks, when after a walk became much worse; croupy	Slight cough, Nov. 4; croupy cough, Nov. 18	Specks on tonsils	Nov. 18

associated with a similar condition of fauces.

Tracheotomy.	Urine.	Result.	Post-mortem examination.		Remarks.
			Resp. fauces.	Resp. larynx.	
No	No note	Death, Oct. 13, apnoea	Tonsils, soft palate, pharynx	Lined with false memb.	
No	Ditto	Death, June 23, apnoea	Tonsils, pharynx	Ditto.	
Mar. 29	No albumen on Mar. 27	Death, Mar. 30	Ditto	Ditto.	
Mar. 1	No note	Death, Mar. 2	No examination, but false membrane extracted from tracheotomy wound.		
No	Albuminuria preceded croup	Death, Feb. 1, apnoea	Pharynx	Lined with false memb.	
No	No note	Death, Oct. 28	Tonsils, soft palate, pharynx	Ditto.	
No	Trace of albumen, Mar. 17 (no albumen, Mar. 18)	Death, Mar. 17, apnoea	Tonsils, uvula	Ditto.	
No	Urine highly albuminous	Death, Dec. 8, apnoea	Soft palate, pharynx, nasal fossæ, stomach	Ditto.	
No	Albuminous, Aug. 11	Death, Aug. 11, apnoea	Tonsils, uvula	Ditto.	
Aug. 11	Highly albuminous, Aug. 11	Death, Aug. 12, preceded by convulsions	Tonsils, soft palate	Ditto.	
Apr. 17	None obtainable; probably scanty. Highly albuminous after death	Death, Apr. 18	Tonsils, uvula	Ditto	False membrane expectorated after tracheotomy.
Sept. 5	Slightly albuminous just before death only	Death, Sept. 7	Tonsils, distinct false membrane	Ditto.	
Nov. 20	No note (kidneys natural)	Death, Nov. 21	Tonsils, root of tongue, distinct false membrane	Ditto	False membrane expectorated after tracheotomy.

156 REPORT OF THE COMMITTEE ON THE RELATIONS OF

No.	Date of admission.	Ætiology.	First symptom of diphtheria.	Membrane seen on fauces.	Laryngeal symptoms.
14	Mar. 13, 1865	No data	Sore throat, difficult breathing, Mar. 10	Tonsils, pharynx	Not marked until Mar. 14
15	Apr. 7, 1864	Contracted in hospital. Scarlet fever, Mar. 17; renal dropsy, Apr. 1	Laryngeal dyspnoea, Apr. 15	Nothing seen	Apr. 15
16	Jan. 24, 1866	Measles, rash, Jan. 16	Croupy cough, Jan. 23	White patch on right tonsil	From the first
17	Nov. 4, 1866	Attributed to cold after moving; subject to sore throat and hoarseness	Lassitude, Oct. 31; sore throat, Nov. 1	Tonsils and uvula covered with false membrane Nov. 3	Hoarse breathing, Nov. 3
18	Jan. 4, 1866	No data	Croupy cough, Dec. 27, 1865	No false membrane seen	From the first
19	Mar. 25, 1868	Old caries of spine; scrofulous child	Slight cough, Mar. 23	Not examined	Husky breathing, Mar. 28
20	Oct. 14, 1868	No data; cough, Oct. 7	Short breath, Oct. 12	Tonsils, false membrane	Oct. 12?
21	Aug. 4, 1868	No data	Lassitude, Aug. 1; lost voice, Aug. 2	No note	Aug. 2
22	Apr. 28, 1869	No data; "cold," Apr. 18	Dyspnoea, Apr. 25	Ditto	Apr. 25
23	Oct. 6, 1868	No data	Coryza, Oct. 2	Nothing seen	Oct. 4
24	Dec. 5, 1868	No data; occasional hoarseness, 6 days	Dyspnoea, Dec. 2	No note	"Laryngeal" breathing, Dec. 4
25	Nov. 3, ?	No data	No data	False membrane seen on tonsils	"Laryngeal symptoms," Nov. 3
26	Nov. 4, 1861	"Wheezing at chest," 14 days; cousin said to be suffering from croup; children met 5 days before, Nov. 2	Breathing noisy and difficult, Nov. 2	False membrane, tonsils, uvula, soft palate	Nov. 2
27	Mar. 7, 1861	No data	Febrile, March 2; dyspnoea, Mar. 3	Fauces, uvula, tonsils	Mar. 3
28	Aug. 12, 1862	Ditto	Loss of voice, Aug. 10	Tonsils, false membrane	From first
29	July 30, 1862	Ditto	"Roughness in breathing," July 26	No note	Ditto
30	Sept. 9, 1862	Child in same house died of "croup" on Aug. 30. Child in opposite house died of "croup" on Sept. 2	Difficult breathing, Sept. 5	Tonsils, uvula, soft palate, pharynx	Ditto

MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX VII. 157

Tracheotomy.	Urine.	Result.	Post-mortem examination.		Remarks.
			Resp. fauces.	Resp. larynx.	
No	Notably albuminous	Death, Mar. 15	Tonsils, soft palate, posterior nares	Lined with false memb.	A wretched rickety child. False membrane repeatedly expectorated.
No	Albuminuria preceded croup	Death, Apr. 17, apnoea	Soft palate, posterior nares	Ditto.	
No	Urine not obtained	Death, Jan. 25, asthenia	Right tonsil, patch of false membrane	Ditto	
Nov. 5	Nov. 5, no albumen; Nov. 6, slight albumen; Nov. 7—11, abundant albumen	Death, Nov. 11	No false membrane	No false membrane	
No	Albumen very abundant	Death, Jan. 5, asthenia	Left tonsil, arytenoid, epiglottic folds, tough false memb.	Lined with false memb.	
No	No note	Death, Mar. 29, apnoea	Uvula, pharynx	Ditto.	
No	No urine obtained	Death, Oct. 14, asthenia	Tonsils, thick false membrane	Ditto.	
Aug. 4	Slightly albuminous	Death, Aug. 5	Tonsils, uvula, soft palate	Ditto.	
No	No note	Death, Apr. 28	Tonsils, soft palate	Ditto	
Oct. 6	Albuminous	Death, Oct. 10	"A little false membrane on tonsils"	Ditto.	Lobar pneumonia right lung
Dec. 5	No albumen in urine	Death, Dec. 15	"False membrane on tonsils"	Ditto.	
Nov. 3	No albumen	Death, Nov. 4	No false membrane	Ditto.	
No	No note	Death, Nov. 5, apnoea	Tonsils, uvula, soft palate, posterior nares	Ditto.	
Mar. 7	Ditto	Death, Mar. 8, apnoea	Tonsils, false membrane	Ditto.	
No	Ditto	Death, Aug. 16, apnoea	False membrane both tonsils	Ditto.	
July 30	Ditto	Death, July 31, apnoea	Tough exudation on tonsils	Ditto	
Sept. 9	A little albumen	Recovery	—	—	

False membrane expectorated after tracheotomy. No sequelæ, Feb., 1863, was quite well.

158 REPORT OF THE COMMITTEE ON THE RELATIONS OF

No.	Date of admission.	Ætiology.	First symptom of diphtheria.	Membrane seen on fauces.	Laryngeal symptoms.
31	June 19, 1862	Brother died of diphtheria June 18. Mother, false membrane on tonsils and soft palate, June. 20. Recovered	Lassitude, June 13	Tonsils, June 14	Laryngeal symptoms, June 22
32	Aug. 29, 1862	No data	Lassitude, Aug. 27; cough, Aug. 29	Tonsils, fauces, Aug. 29	Aug. 29
33	Aug. 11, 1863	No diphtheria known in neighbourhood	Throat sore, July 21; dyspnoea, Aug. 9	Tonsils	Aug. 9
34	July 20, 1863	One child died 2 months ago of "diphtheria"	Lassitude, July 15; croupy cough, July 18	Ditto	July 18
35	Apr. 19, 1863	No diphtheria known in house	Lassitude, Apr. 10; cough; dyspnoea, Apr. 16	Tonsils, patch of exudation on both	Croupy cough, Apr. 18
36	June 25, 1863	No data	Cough, June 15; dyspnoea, June 22	No note	Breathing hoarse, June 22
37	Feb. 19, 1863	No diphtheria known in neighbourhood	Coryza, Feb. 17	Tonsils, soft palate, pharynx	Croupy cough, Feb. 18
38	June 15, 1864	Ditto	Lassitude, June 8; sore throat, June 12	Tonsils, thick exudation	Husky voice, June 15
39	July 13, 1864	No data	"Throat swollen," July 7	Tonsils, exudation	Croupy cough, July 8
40	Aug. 31, 1865	Ditto	Throat sore, Aug. 30	Fauces covered with false membrane	None
41	June 6, 1868	Ditto	Lassitude, May 31; sore throat, June 3	Tonsils and posterior pharynx	Dyspnoea, June 5
42	June 29, 1868	Ditto	Febrile, June 19; neck swollen, June 20	Tonsils, false membrane	Husky breathing, June 26

MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX VII. 159

Tracheotomy.	Urine.	Result.	Post-mortem examination.		Remarks.
			Resp. fauces.	Resp. larynx.	
No	Very albuminous	Death, June 23, apnoea	- No post mortem		Private patient.
Sept. 1	Ditto	Death, Sept. 3, apnoea	Tonsils, pharynx, false membrane	Lined with false memb.	
No	No note	Death, Aug. 13, asthenia	Tonsils, uvula, soft palate, false membrane	Ditto.	False membrane expectorated after tracheotomy.
July 21	Highly albuminous	Death, July 28	Tonsils, soft palate, false membrane	Ditto	
Apr. 19	Very albuminous	Death, Apr. 22	No false membrane	Ditto.	
June 25	Decidedly albuminous	Death, June 26	Tough exudation on each tonsil	Ditto.	
Feb. 20	Urine albuminous after death	Death, Feb. 20	Tonsils, soft palate, pharynx, false membrane	Ditto.	
June 16	Notably albuminous	Death, June 18	No false membrane	Ditto.	
July 13	Ditto	Death, July 14	Tonsils, uvula, false membrane	Ditto.	
No	None obtained	Death, Sept. 1	Tonsils, soft palate, false membrane	No false membrane in rima glottidis, not much below arytenoids.	
June 6	Very albuminous	Death, June 6, apnoea	Tonsils, soft palate, pharynx, false membrane	Lined with false memb.	
July 1	Highly albuminous	Death, July 1	Tonsils, false membrane	Ditto.	

CLASS 2.—Cases of Membranous Laryngitis

No.	Date of admission.	Ætiology.	First symptom of disease.	Membrane seen on fauces.	Laryngeal symptoms.
1	Mar. 3, 1863	No data	Laryngeal	None	Hoarse, Mar. 2
2	Nov. 2, 1864	Oct. 29, exposed in street to a cold wind; became hoarse same night	Ditto	Ditto	Hoarse, Oct. 29
3	Oct. 21, 1864	No data	Ditto	Ditto	Hoarse, Oct. 9
4	Nov. 17, 1865	Ditto	Cough, 3 weeks; hoarse, Nov. 12	Ditto	Hoarse, Nov. 12
5	Sept. 20, 1865	Whooping-cough for two months before croup. Child ill in house with sore throat (supposed scarlet fever)	Laryngeal	None seen during life	Hoarse, Sept. 19
6	Private patient	Scarlet fever in house	Lassitude, Mar. 14; sore throat	Ditto	Croupy cough, Mar. 19
7	Sept. 10, 1861	No data. Croup occurred while in hospital for dropsy, probably scarlatinal	Feverish, Sept. 8; dropsy, Sept. 9 (albuminuria)	Ditto	"Tracheal breathing," Sept. 12
8	Sept. 19, 1861	Scarlet fever, Aug. 29; dropsy, Sept. 12	—	Ditto	"Tracheal breathing," Oct. 8
9	Jan. 30, 1861	In hospital for spina bifida	Laryngeal cough, Mar. 7	Ditto	Laryngeal cough
10	May 9, 1862	Subject to laryngismus stridulus; rickets	"Stridulous breathing," May 7	Ditto	Stridulous breathing
11	May 23, 1862	"Tracheitis," with symptoms of croup; in hospital from Mar. 4 to Mar. 23, 1862; went out well, and kept so until May 20	Feverish, May 20; headache, delirium. No croupy symptoms noted before admission	Ditto	Breathing tracheal, May 23
12	Feb. 20, 1862	Croup supervened in hospital on 12th day of typhoid fever	Throat sore, Feb. 27; hoarse breathing	Natural	Hoarse breathing, Feb. 27
13	Mar. 3, 1863	Supposed to have caught cold on Mar. 1. Went to a warm church, and then had half an hour's walk home in the cold; forgot to fasten cloak. No diphtheria in neighbourhood	Hoarse voice, same evening; violent, Mar. 1	None	From the first

MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX VII. 161

not associated with a similar condition of fauces.

Tracheotomy.	Urine.	Result.	Post-mortem examination.		Remarks.
			Resp. fauces.	Resp. larynx.	
Mar. 3	Albuminuria	Death, Mar. 19	Natural	Filled with false membrane.	
No	Urine not obtained	Death, Nov. 3	Ditto	Ditto.	
Oct. 21	Albuminous	Recovery	—	—	False memb. expectorated after tracheotomy.
Nov. 17	None obtained	Death, Nov. 18	Natural	Ditto.	
Sept. 20	Notably albuminous	Death, Oct. 2	No post mortem	—	False memb. expectorated after tracheotomy.
Mar. 21	No note	Death, Mar. 23	Natural	Filled with false membrane.	
No	(Renal dropsy)	Death, Sept. 15	Ditto	No true false membrane, but a thin layer of thick mucus in air-tubes	No pleural effusion noted.
No	Ditto	Death, Oct. 10	Ditto	Lined with false membrane	False memb. expectorated, Oct. 10.
No	No note	Death, Mar. 9	Red and congested	Ditto.	
No	Ditto	Death, May 9	Natural	Ditto.	
May 24	No albumen	Death, May 27	Ditto	Ditto.	
No	Ditto	Death, Mar. 1	Tonsils "ulcerated;" no false memb.	Ditto.	
Mar. 3	Albumen appeared Mar. 9, and remained till death	Death, Mar. 19	Natural	Broken-up exudation.	

162 REPORT OF THE COMMITTEE ON THE RELATIONS OF

No.	Date of admission.	Ætiology.	First symptom of disease.	Membrane seen on fauces.	Laryngeal symptoms
14	Oct. 28, 1862	Acquired in hospital. Under treatment for pneumonia	Nasal discharge, Nov. 7	None	"Croupy" cough Nov. 12
15	Nov. 16, 1863		Seemed to have a bad cold, Nov. 9	Ditto	Croupy breathing, Nov. 15
16	Nov. 1, 1865		Cough, 1 week; voice hoarse, Oct. 29	Ditto	Hoarseness, Oct. 29
17	Apr. 5, 1866	Ditto	Hoarseness, Apr. 4	Ditto	Hoarseness, Apr. 4
18	Apr. 8, 1867	Measles rash, Mar. 27	Dyspnœa, Apr. 2	—	From Apr. 2
19	June 20, 1867	No data	No data	None	Present on admission
20	Apr. 17, 1868	"Croup," 2 years ago, several attacks since	Febrile, Apr. 2	Ditto	Dyspnœa, Apr. 5
21	Apr. 2, 1870	No data	Noisy breathing, Mar. 30	Ditto	From Mar. 30

MEMBRANOUS CROUP AND DIPHTHERIA—APPENDIX VII. 163

Tracheotomy.	Urine.	Result.	Post-mortem examination.		Remarks.
			Resp. fauces.	Resp. larynx.	
No	Albumen appeared Nov. 14	Death, Nov. 19	Natural	Broken-up exudation	Double pneumonia.
Nov. 19	Slight albumen, Nov. 19	Recovery	—	—	A little false memb. expectorated.
Nov. 1	Slight albumen, Nov. 8	Death, Nov. 3	No post mortem examination	False membrane in trachea; larynx not examined	False memb. expectorated after tracheotomy.
No	No albumen	Death, Apr. 7	No false membrane	False membrane below true cords.	
No	Ditto	Death, Apr. 13	Natural	Lined with false membrane.	
No	No note	Death, June 25	No note	Ditto.	
Apr. 17	No albumen on Apr. 18	Death, Apr. 24	Natural	Ditto (thick false membrane)	Pneumonia.
Apr. 9	No albumen on Apr. 4	Death, Apr. 10	—	Larynx much reddened; "what seemed to be soft exudation" in trachea.	

CLASS I.—*False membrane in larynx and fauces.*

Sex.—Males, 18. Females, 23. Not said, 1 = 42 cases.

<i>Age.</i> —From 1 to 2 years.	2—3.	3—4.	4—5.	5—6.	6.		
Males . 1	7	5	1	2	2	0	= 18
Females 3	7	5	4	3	0	1	= 23
Not said = 1							1
							42

Time of year.—Month of admission to hospital:

Jan.	Feb.	Mar.	April.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.	
3	2	5	4	0	6	4	6	3	3	4	2	= 42

Ætiology:

Other cases of diphtheria in neighbourhood	= 7	} 12
Contracted in hospital	= 5	
Supposed due to catching cold	= 3	
No data	= 27	

Earliest local symptom of diphtheria:

Sore throat	14
Croupy cough, hoarseness, or dyspnoea	23
Sore throat and laryngeal symptoms simultaneous	2
Coryza	2
No data	

Time of onset of laryngeal symptoms:

Day of disease	1	2	3	4	5	6	7	No data.
Cases	16	4	4	3	3	1	1	5

Tracheotomy.—23 times and 1 recovery.

<i>Albuminuria.</i> —Old nephritis before croup	2
Present during life	20
Urine not obtained during life; albuminuria after death	2
Not albuminous so often as examined	3
No data	15

Results.—Died . . 18 males. 22 females. 1 unknown sex = 41 deaths.
Recovered . 0 „ 1 „ 0 „ = 1 recovery.

CLASS II.—*False membrane in larynx; not in fauces.*

Sex.—Males, 10. Females, 11 = 21 cases.

<i>Age.</i> —From 1 to 2.	2—3.	3—4.	4—5.	5—6.	6.	7.	11.	
Males . 1	3	3	0	2	0	0	1	= 10
Females 1	0	1	4	3	1	1	0	= 11
								<hr/> 21

Time of year.—Admission:

Jan.	Feb.	Mar.	April.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.	
1	1	3	4	2	1	0	0	3	2	4	0	= 21

Ætiology:

Attributed to exposure to cold = 2 No data = 8
 Contracted in hospital . . = 4
 Diphtheria in neighbourhood . = none said to be so.
 Second attack of "croup" in 2. During measles=1. During hooping cough=1
 During scarlatinal dropsy=2. During typhoid fever=1. Scarlet fever in
 house=2.

Earliest local symptom of disease:

Sore throat 1
 Laryngeal 17
 Both at same time 1

Coryza 1
 No data 1

Time of onset of laryngeal symptoms:

Day of disease 1 4 6 Doubtful.
 Cases 15 2 2 2

Tracheotomy.—11 times and 2 recoveries.

Albuminuria.—Old nephritis before croup 2
 Present during life 7

Not present so often as examined 6
 No data 6

Results.—Died 19
 Recovered 2, both females.

APPENDIX VIII.

REPORTS TO THE MEDICAL OFFICERS OF THE PRIVY COUNCIL AND LOCAL GOVERNMENT BOARD.

THE following is a list of the principal Reports made to the Medical Officers of the Privy Council and Local Government Board with reference to outbreaks of diphtheria, or having some bearing upon the subject. Through the kindness of Dr. Buchanan the Committee have had access to these reports, and reference has been made to them upon several important points in the history of diphtheria.

It was intended to append a digest of these reports to the Report of the Committee, but as the greater part of them have been published, it is thought that it will be sufficient to give a list of the more important for purposes of reference, and that this will be of value in future work upon the subject. The majority of these reports have, moreover, no special reference to the relations of diphtheria and croup, the facts brought out in them upon this point arising only incidentally.

LIST OF REPORTS.

- 1860.—Second Report of the Medical Officer of the Privy Council Reports of Dr. GREENHOW and Dr. SANDERSON on Diphtheria.
- 1871.—Dr. HOME, 'Outbreak of Diphtheria at Newton Valence, Hampshire.'
- Dr. GWYNNE HARRIES, 'Diphtheria in Porlock, Luccombe, and Selworthy.'
- 1872.—Dr. HOME, 'Diphtheria in Charles Registration Sub-district of Plymouth.'
- Dr. GWYNNE HARRIES, 'Sanitary Arrangements of Villages in Worksop Union.'
- Dr. AIRY, 'Diphtheria in Longbenton Subdistrict.'
- Dr. THORNE THORNE, 'Outbreak of Diphtheria at Andover.'
- Dr. THORNE THORNE, 'Diphtheria at Great Milton (Thame Union).'

- 1873.—Dr. GWYNNE HARRIES, 'Sanitary Condition of Guisborough (Enteric Fever).'
- Dr. THORNE THORNE, 'Sanitary Condition of Guisborough (Diphtheria in Lofthouse Subdistrict).'
- 1876.—Mr. W. H. POWER, 'Diphtheria at Brailes, Warwickshire.'
- Dr. AIRY, 'Diphtheria at Penkridge in Staffordshire.'
- 1877.—Dr. AIRY, 'Sanitary State of Ludlow Registration District.'
- Dr. THORNE THORNE, 'Epidemic of Diphtheria at Great Coggeshall, Essex.'
- 1878.—Dr. THORNE THORNE, 'Prevalence of Diphtheria in Urban Sanitary District of Denbigh.'
- Dr. THORNE THORNE, 'Diphtheria at Llanrhaid.'

A CASE OF MORPHŒA.

BY

GEORGE GASKOIN,

SURGEON TO THE BRITISH HOSPITAL FOR DISEASES OF THE SKIN.

Received March 26th—Read November 12th, 1878.

THE opinion that morphœa and leprosy are dissimilar and wholly different diseases is one that seems to be recommending itself to certain minds in the profession however much discountenanced by the teaching of earlier writers. There are cases and circumstances, indeed, which favour the idea of their distinctness, and others, again, which lead to quite an opposite conclusion. It is a question of no modern date, but descending to us from the past.

To multiply discussion on such a topic can lead to little good, the solution can only come from earnest clinical inquiry. The undetermined state of this question, it may fairly be presumed, will lend an additional interest to the details of the case that follows.

Harriet J—, æt. 24, unmarried, and of purely English nurture and extraction, presents herself with certain nodular masses which lie on the right side of the lower jaw. They are elongated, and four-sided in figure; their surface displays a bony or ivory whiteness, it is scantily marked by venules, which in vast numbers surround their margins and give colour to the parts investing them. Beyond the redness which is thence derived, there is but little attendant dark stain. They appear to be raised or lifted up by a certain amount of

subcutaneous deposit. The size of the individual masses is very much that of the terminal phalanx of the adult little finger, but varying somewhat in this respect. It may be said that they answer perfectly well to the description of *morphœa alba tuberosa* from the hand of Erasmus Wilson, the *dartre de graisse* of Retz. These eminences or nodules are five in number, and they form a double group, three of them lying near the angle and ramus of the jaw, and two others along the body of the bone at its lower edge extending to the symphysis. At the present time they are in some degree contorted or twisted on themselves. They may be said to exceed the usual dimensions of this kind of morphœa. Our attention is especially engaged by a retraction or contraction of the skin immediately adjacent to them, which in some parts takes the form of an elongated scar. This is seen in each group; in that which lies anteriorly and in a line with the jaw it would seem as if, at some previous date, a strong knife had been used to incise the bone to the extent of one inch in direction of its length, the result being left in a cicatrix. The effect of the retracted and puckered skin is such that the nodules are thrown into higher relief, and, as before said, somewhat displaced or contorted. I cannot but observe in this subcutaneous contraction a certain similarity to leprosy.

I have commenced by drawing attention to these nodules on the face because it is here that we find the disease in the greatest activity, though elsewhere it is seen in more ample proportions.

Let us now examine the flat of the temple on the same side. There is here something like *alopecia areata* when very far advanced in the stage of recovery, that is to say, some part of the hair is stunted and short, while more of it is pretty long, and, indeed, nearly as much so as the rest of the hair, with which it contrasts in colour, as displaying a yellow shade. It streaks the dark brown tresses by its threads of gold. It thus becomes an object of minute attention in arrangement of the head dress. Corresponding to this golden band of hair there is found on the skin of the

part a well-marked yellow stain, not deep in tint, but obvious enough.

In this region we have some cause to think the disease first had its commencement; in this spot, as we are informed, it first became apparent to her friends. For some two years or more the right temple was occupied by tubercles, which do not seem at any time to have been followed by a deeper stain than that which is at present discernible.

If we proceed to draw a line from the temple to the projecting part of the cheek, we shall find it crossed at right angles by a very noticeable scar, which lies directly over the malar protuberance. This resulted from a violent fall and contact with some iron substance, which happened in the patient's childhood, at a time when she had suffered long from very sickly health the consequence of scarlatina.

The tubercles appeared on the temporal region some two years after this blow. A good deal of uncertainty, however, attaches to the early history of the case; it is not easy to fix on any date for the commencement of this disease, nor indeed to decide with much positiveness in what part it first began. It commenced, no doubt, in the years of childhood, and possibly from laceration of nerve. We are also told that in an early period of youth her chest was covered with "suetty masses," and likewise with stains which in course of time became dispersed. We can, at least, not do amiss if we assign thirteen years as the minimum duration of her complaint.

On the left side of the chest at the present time, there are two large patches of morphœa with deposit (*morphœa alba tuberosa*). They are typical, being waxen on the surface, and are more level with the skin than those beneath the jaw; they are large and horizontally extended. Here also the disease is in full activity, and there is more seen of it as we approach the waist. On the greater part of the chest anteriorly the skin is normally white, and the shoulders the same. Above the clavicle on the left side we discern a bright yellow stain, it resembles that on the opposite temple except that it is deeper in hue. By the application of warm

water and sponge I am able to reproduce in its centre the contour of an almond-shaped areola, which is indisputably a stain from antecedent morphœa, not indeed limited by a very clear outline, but rather feeble and diffused.

Hitherto, we have seen, in this patient, the *morphœa alba tuberosa*; we have now to include in our description certain other manifestations which, singly considered as seen in this country, are of rarest occurrence, but which here apparently through the operation of one morbid process seem blended with the former complaint.

If we look at the inside of the left arm about the middle of its length, we find there a large round macula. It is black in colour, orbicular in shape, and defined by a very clear boundary. In diameter it is about three inches and a half, and its situation on the limb is very slightly diagonal. It is soft, pliable and uniform. A number of equi-distant points of white colour are perceptible on its surface, as minute dots upon a black ground. These are the white summits of elevated and erect papillæ which, without break or interruption of any kind, and with a perfectly even distribution pervade its surface. We may moreover remark, that in the neighbourhood of the patch, for some two inches more or less from its margin, the papillæ are in like measure elevated, but are neither black in colour nor with white tips. Of such dark patches as this on the arm, there are others on the lower limbs and trunk. The macula before us is smallest in size and, as compared with the rest, of a somewhat feebler negro tint. There is but this one on the upper limbs.

Corresponding with this upon the arm and perfectly comparable to it, is a larger patch on the opposite thigh. It is seated rather diagonally on the limb, being slightly oval in form and it lies anteriorly, but on the inner side. Beginning just above the right knee it is divided from a yet larger macula, which is seated higher up, by a ribbon of skin with perfectly normal tint, which traverses the thigh anteriorly after the manner of the sartorius muscle, but not in the same direction, and thus appears as a not inelegant white band. The more highly seated of these two maculæ ascends

to the region of the pelvis, where it coalesces with other huge stains of the same dark negro shade. On the left thigh much the same occurs, with the difference that the maculæ only begin high up. These patches whether single or blended, have a clear defining outline, and we may pursue it to a considerable extent in their coalescence posteriorly, as they blend in the pelvic region. Such parts we find invested with a dark black stain, which contrasts with the little that remains in their neighbourhood of normal pigmentation; there is here found no half shade. It is otherwise in the bend of the waist, which is indeed pigmented to excess but without any boundary line; in receding from this part the blackness gradually fades and subsides into the normal tint, quite otherwise than with the maculæ.

Posteriorly a limit is found to the maculation at the upper border of the pelvis, above this point generally a normal colour obtains. An exception to this is found in a lozenge-shaped macula which is placed centrally lengthwise and across. It extends from the cervical to the lumbar regions, being narrow in proportion to its length, and being the only one of these maculæ that is symmetrical. It would appear to be now far advanced in the course of obliteration. In this process it does not exchange the black for any intermediate shade, but it is broken up by a coarse white mottling, which in time will supersede the black.

On this posterior aspect of the body one must also observe some notable spots of leucoderma, one of them as large as a florin, while of two others each has the size of a shilling piece, the latter are united and conjointly take the form of a fig. These are situated near the crest of the ilium on the right side. The leucoderma is somewhat dull and faded, and there is a slight unevenness of the surface.

We have thus seen a considerable part of the body invested with a black colour, while the greater part of the limbs and the left side of the face and head are wholly free from disease. There yet remains something of interest to be said about the hypogastric region, all in this neighbourhood is deeply, but diversely, pigmented. We here observe on a

black ground a multitude of light coloured areolæ. These are of the shape and size of an almond, horizontally extended, perfectly supple and level with the surface without unevenness of any kind. The areolæ vary in colour being more or less of a dirty brown, yellow, or white, they also vary somewhat in size. As we ascend to the region of the waist we come upon the *morphæa alba tuberosa* in greater or less activity, though very sparsely scattered. It can scarce be otherwise than that these almond-shaped areolæ which pervade the lower part of the abdomen are extinct or fading foci of the *morphæa tuberosa*.

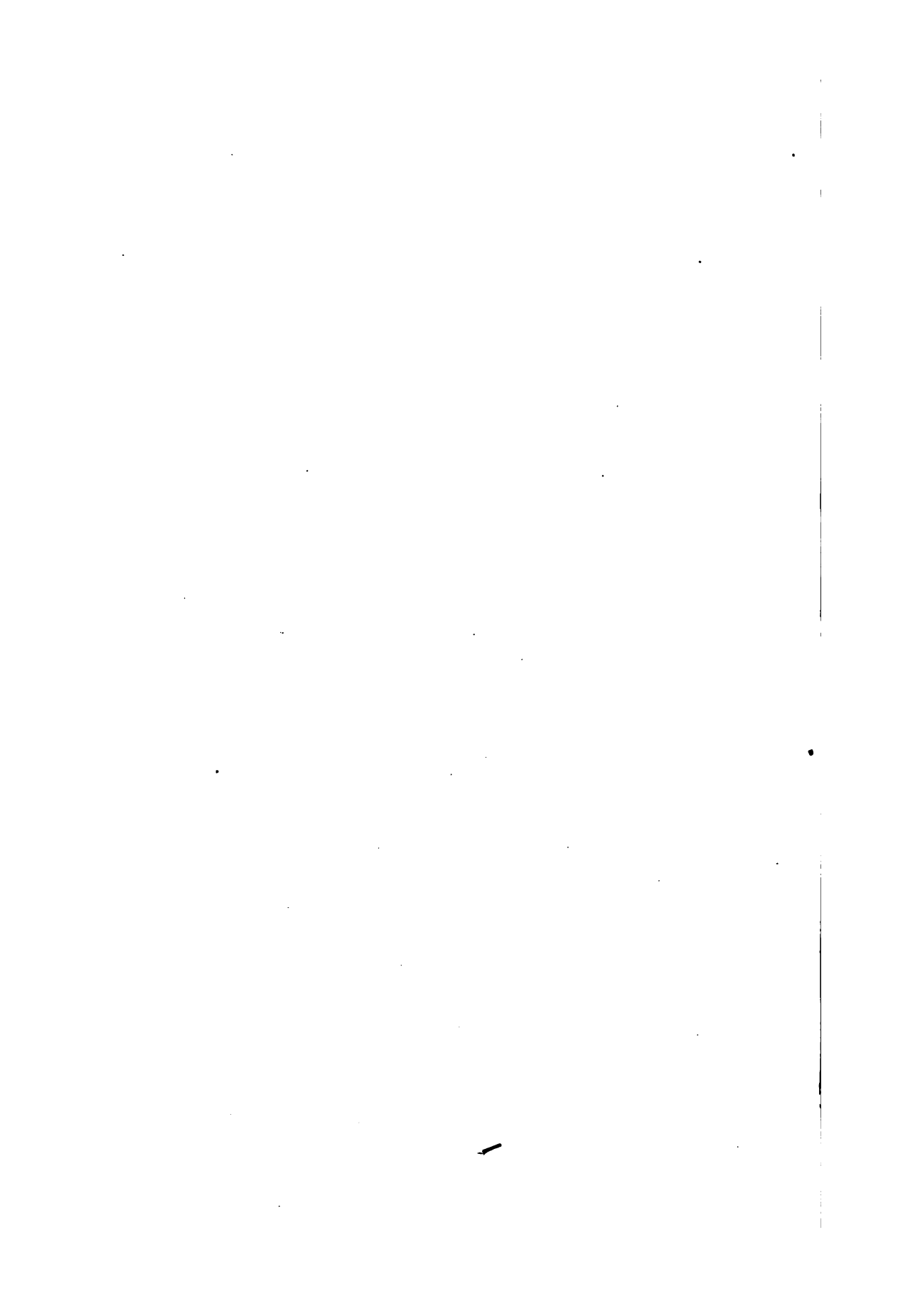
There are no minor manifestations like dark spots or puncta; no ulcerations or pemphigus. Pain, anæsthesia, pruritus, in this patient are scarcely or not at all observed; there is only a trifling degree of local heat at times and twitchings. The patient's health is ever languid and disturbed, but with no especial ailment. Her frame is large and full proportioned, her functions regular. The complexion tends to blonde, the colour of her hair being dark brown, and her eyes of blueish grey. Nails, brows, and hair are strong, the latter even luxuriant. The history of the patient's family reveals very much delicacy and frailty of constitution, which might with some reserve be spoken of as scrophula, but in which abscesses are the most prominent feature, and general debility; but no skin disease has been known among them with the exception of eczema. Her circumstances are moderately advantageous. The treatment throughout has been alterative and sustaining; the prognosis is deemed to be uncertain.

This case might be called *morphæa alba et nigra*. Such would have been its designation in an earlier period of medical science. Both one and the other were *species famosæ*, and reckoned as of a "bad sort," that is to say, dangerous, of an uncertain event and also akin to leprosy. The dark is sometimes mentioned as the worst; but enlarged dimensions even in the middle ages were also an argument of much force toward such conclusion. In its original signification it is not improbable that morphea signified a

dark-coloured stain ;¹ but of late there has been a disposition to confine the term to such of its manifestations as are white. My own experience of *morphœa alba* whether in the tuberoso or atrophic variety, instructs me that it is often accompanied by additions of pigment and especially by mulberry stains as well as by leucoderma. I have seen nothing, however, comparable to the darker maculæ which are described in the preceding pages, except as a symptom of leprosy, and then according to my individual experience not so dark as these, though otherwise similar. They are perfectly described, however, in works which treat of leprosy, especially in the West Indies and western hemisphere, and as not being altogether absent, though rare in that of northern Europe.² I would, however, refrain from expressing an opinion as to the identity of morphœa and leprosy.

¹ See Hensler, 'Vom abendländischen Auss tze,' . 42, A.D. 1790.

² See Hensler, op. cit., pp. 251, 254.



ON THE
CONDITION OF THE SKIN IN TINEA
TONSURANS.

BY

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(Received May 11th—Read November 12th, 1878.)

IN a paper read at a recent meeting of the Medical and Chirurgical Society, Dr. Thin¹ described the condition of the skin in Tinea tonsurans. His specimens were taken from the horse, but at the same time I was myself engaged in the examination of the disease in the human scalp, and some of the results I stated to the meeting. I believe that Dr. Thin's is the first published account of an actual microscopic examination of the affected skin in this disease, the statements made by various authorities as to the localisation of the fungus being, apparently, founded on examination during life of epidermis detached from the surface, and hairs extracted from the follicles. Whether examinations of the deeper parts of the skin and of the hairs *in situ* have been made or not, it does not appear that any appeal is made to them by those writers on Tinea tonsurans who have discussed the manner of invasion of the hairs of the fungus; and I, therefore,

¹ "On the Condition of the Skin in Tinea Tonsurans," 'Med. Chir. Trans.,' vol. lxi, 1878, p. 179.

offer the present communication as a contribution to our knowledge on the subject.

When the opportunity, which I had long been waiting for, at last presented itself to me of investigating this matter, I had in view to ascertain whether the fungus was confined to the hair itself, and if not, how far it spread into the structure of the root-sheaths and hair-follicles; because it seemed that the deeper it extended, the less could one expect the various methods of treatment, by epilation or by parasiticides to be quickly successful.

On this point Dr. Thin's specimens from the disease in the horse confirmed the observations of Küchenmeister. The latter says¹ that the fungus is found in the substance of the hair itself, that it is never found between the cells of the epidermis and never in the scales which cover the head. Kaposi,² however, describes the *Trichophyton* as being found in the hairs and *their root-sheaths*, as well as in the layers of the epidermis in the vesicular and squamous forms of the disease.

The subject of the disease in the present case was a child aged 5 years, who died in Guy's Hospital from a large cheesy tubercle occupying the right half of the cerebellum, and invading the right side of the pons. The symptoms had first shown themselves seventeen months previously, and the child had been under my care in the hospital ten months. During the whole of this time he became steadily worse, and during the last six or eight weeks was only semi-conscious, fed with difficulty, and rapidly wasting. The ringworm affected a circular patch about two inches in diameter on the left side of the head over the parietal bone, and other smaller patches were scattered over the head. When first seen, there were numerous short broken hairs, and abundant desquamation of the epidermis in small branny scales; but there was no redness or tenderness. Very little hope was entertained on account of the intracranial disease, that any treatment would be successful, while the ultimate prognosis of the case was so

¹ 'Manual of Parasites,' vol. ii, p. 141, Syd. Soc., 1856.

² Hebra und Kaposi's 'Hautkrankheiten,' Theil ii, p. 638.

certainly fatal, that we could have none but a scientific interest in the cure. However, in May and June, an ointment of goa powder, and one of petroleum were from time to time applied. The former caused the usual erythematous redness, but no effect on the disease itself was observed, and at the end of July, the hairs extracted were still found to be loaded with spores. For the few remaining months of his life, during which the fatal symptoms made rapid progress, little attention was paid to the ringworm, though the hair was kept close cut for purposes of cleanliness. The boy died on December 14th, 1877.

After death a large portion of skin was removed from the left side of the head, and placed in a mixture of equal parts of alcohol and solution of chromic acid of about one per cent. strength. During February I commenced to work upon it, and made a great number of sections parallel to the course of the hairs, parallel to the surface of the skin, and in other directions. Some were stained in logwood, but by far the greater number in picro-carmin. In the latter sections it is not difficult to detect the affected hairs with a low power. The tissues of the skin, generally, take up the carmin in the usual way, but the horny tissues are stained by the picric acid alone; thus the cutis proper and the mucous layer of the epidermis are stained pink, as are also the corresponding hair-follicles, papilla, and outer root-sheath; but the horny layer of the epidermis becomes yellow; the hairs are yellow when small, and yellowish green or deep green when thick, or pigmented; while the inner root-sheath is variously coloured, very pale pink in the neighbourhood of the papilla, colourless or yellow in the rest of the follicle, with blotchings of deep pink, especially just where the hair swells out into the bulb. Where the hair is extensively diseased or utterly destroyed by the presence of numbers of spores it presents an orange-brown or reddish-brown colour, by which it is readily distinguished from the healthy green and yellow hairs; in thinner sections it has a paler brown colour.

Hairs and portions of hairs, are, however, often found in

which the disease is less advanced ; these are still in a position to assume a greenish colour and they are generally only recognised as diseased on more careful examination with higher powers.

What is then seen is that the shaft of the hair contains several fine filaments (or tubes) running parallel to the hair and to one another. I need not describe these further ; the appearances of the filaments themselves are, of course, not different from what may be seen in hairs extracted from the living subject. When the process of growth is more advanced and luxuriant, a high power shows that the hair substance is almost entirely destroyed. The space within the internal root-sheath has lost its uniform outline and is irregular, though not, as far as I have seen, to any great degree ; and this space is filled with the spores of the fungus. In the middle, and, as it were, separated by them from the sheath with which it ought to be in contact, lies the remnant of the hair. It has also the irregular outline, the opaque appearance and the fibrillated structure, with which we are familiar in hairs removed during life (Pl. I, fig. 1, and Pl. II, fig. 4),

In seeking out the limits to the growth of the fungus, I found some difficulty in determining the extent to which it spread in a downward direction towards the root ; as out of a large number of sections, only a few have fallen in such a way as to illustrate this point of the inquiry. I have never observed the soft tissue constituting the bulb of the hair to be completely involved, but in some sections I have seen mycelium threads extending as far as the point at which the shaft begins to swell out into the bulbous portion. In these the outline of the hair was still preserved, and though filled with threads it was not as yet obscured by separate spores (Pl. I, figs. 2 and 3).

I never found the fungus to extend below this point.

The difficulty of getting diagrammatic sections of hair, bulb, and papilla, is still greater ; and, although some of my slides show such sections, in these the hairs are healthy, and I have, unfortunately, not met with any diseased hairs,

cut exactly in the way indicated. On the other hand, I have examined numbers of papillæ, isolated, or in connection with short stretches of hair; and even in portions of the scalp much affected with the disease, I have never found them anything else than perfectly normal.

With respect to the extension of the growth in a lateral direction, this is clear that in none of my specimens are there any spores or mycelium threads in the outer root-sheath, which corresponds to the mucous layer of the epidermis; the cells of this layer are quite normal (Pl. I and II, figs. 1, 2, 3, 4). To determine the condition of the internal root-sheath requires careful examination. As I have said, in the advanced stage, the hair is both occupied and surrounded by spores, so that the latter come into actual contact with the internal surface of the internal root-sheath; and, in exceptional cases this root-sheath is so far obscured by them, that it would be impossible to say it was not involved. But the rule is for the inner root-sheath to be easily discernible, whether high up in the follicle near the surface, or low down near the papilla; whether the hair be in the condition just described, or only invaded by mycelium threads in longitudinal arrangement. It is remarkably well shown in Pl. I, figs. 2 and 3, how, in the early stage, the threads confine themselves entirely to the hair, without any attempt to encroach upon the tissues beyond; and Pl. II, fig. 4, shows the integrity of the inner root-sheath in the more advanced conditions.

As might be expected after this, the structure of the follicle itself, the cutis, and the subcutaneous tissue in which the follicle is embedded, show no signs of the parasite.

It only remains to speak of the epidermis. The horny layer can be made the subject of examination during life, and, perhaps, better than after death, as it is very likely to be loosened or detached by the fluid employed to harden the skin itself. I have succeeded, however, in retaining a considerable quantity of this layer, some of it in the immediate neighbourhood of diseased hairs; but though the presence of spores or threads in the horny layer would be particularly easy to detect, I have failed utterly to find them.

Since it is possible that the most superficial layers have been removed post mortem, the present specimens would scarcely justify me in saying the horny layer was never implicated. But it is remarkable how much of the horny layer of the epidermis may be completely free from infection even in close proximity to a diseased hair (Pl. II, fig. 4).

The deeper layer of the epidermis, like the outer root-sheath is never involved.

It will be seen, therefore, that these observations are in favour of the statements of Küchenmeister and opposed to those which Kaposi advances in reference to the implication of the root-sheaths. The hair-follicle, papilla and root-sheaths, the cutis, subcutaneous tissue, and mucous layer of the epidermis, are entirely free, while the only parts invaded are the hair proper, and perhaps the horny layer to a slight and superficial extent.

Of the breaking and twisting of hairs described by Dr. Thin, as well as the inflammatory changes, I have seen but little. The former condition may have been present at some or other part of the diseased patch, but in all the diseased hairs which were cut in sufficient lengths for such an observation, the straight course of the follicle was maintained, and no such fracture or twisting was seen. Only very rarely was I able to find any traces of inflammatory action. Here and there was a spot where the tissue was filled with young cells, but for the most part the whole tissue was quite free from cell-growth or vascular changes even in the immediate neighbourhood of the follicles of diseased hairs.

This need not be considered as contradicting Dr. Thin's observations; for, firstly, in the weakened condition of the child during the last months of its life, the hairs might grow with less vigour, and thus, more gently, and without distortion, expel the separated fragment; and, secondly, if the same cause were not sufficient to account for active irritative changes, it is clinically well known to us that the more chronic stages of *Tinea tonsurans* are unaccompanied by inflammation; and the disease in this case had lasted more than nine months.

From the results of the above investigation, I think one may infer that the mode of invasion of the disease is somewhat as follows :—Whether or not the fungus first takes root in the horny layer of the epidermis, the first point at which the hair is attacked is probably that portion of the shaft immediately on a level with the surface of the skin. There I should suppose the fungus grows well into the substance of the hair and spreads by the advance of mycelium threads certainly downwards towards the papilla, and perhaps also upwards into the free part of the shaft. Then these three changes must be going on simultaneously ; (a) the complete destruction of the hair at the part first invaded ; (b) the advance of the threads into the hitherto healthy shaft or root, and (c) the natural growth of the hair in an upward direction as it is supplied from the papilla. By this last action, the portion of the hair which is first attacked and most thoroughly diseased, is carried beyond the level of the skin, and thus, losing at the same time, both cohesion and support, the hair yields, and is removed by the usual daily accidents of friction by combing, brushing, &c.

The spread of the fungus in each follicle is necessarily measured by the growth of the hair, since we have seen that it is confined to the substance of the hair itself and does not extend to the other tissues. One cannot help recognising the essential difference between what the fungus invades and what it leaves untouched ; between the living root-sheath, follicle, and papilla, on the one hand, and the relatively dead hair-substance on the other. Dr. Thin, also, has remarked on the preference of the fungus for the most dead tissues such as the hair and the horny layer of the epidermis ; and with this fact in view one can see that the plant spreading down the hair would never be able to approach within a certain distance of the papilla, because of the formative power of the immediately adjacent hair-cells, which would enable them to resist its inroads, until, in their turn, they had assumed the dense, dry, dead condition of the more remote parts of the hair ; and should the papilla cease to form any fresh hair, the multiplication of the fungus spores in that follicle would be

necessarily brought to a stand-still for want of their own proper nutriment.

There remains the question whether one can from these results derive any suggestions as to methods of treatment or the operation of remedies in this disease. The local measures usually employed are of three kinds ; the practice of epilation, the application of irritants, and the use of so-called parasitocides. The difficulty presented by the last method is, that of ensuring the penetration of the agent to the deepest part of the follicle, for it is there, I take it, that the most active growth is going on. Though there may be thousands of spores in the rest of the follicle they have by this time exhausted their supply of nutriment, and it is in the deepest part only that is found the material for the further development of the fungus. Epilation labours under a somewhat similar disadvantage, namely, that an infected but not exhausted portion of the hair is apt to be left in the follicle. In a healthy hair, the weakest point is at the connection of the bulb with the papilla, where the cells are soft, and by steady traction the hair may be separated ; but, in one infiltrated with fungus the connection of the bulb with the papilla is relatively strong, and the portion which holds only the advancing threads of mycelium has more cohesion than that which has contributed to the development of crowds of sporules. Hence the hair will yield at this last point instead of at its junction with the formative papilla.

With regard to the action of irritants, many of which are of great service in curing the disease, Dr. Thin has offered an explanation which is not altogether improbable, namely, that the process of inflammation induced by their use, results in a persistent effusion of serum, which penetrating the hair-sheaths, and bathing, unceasingly, the spores that surround and infiltrate the hairs, acts destructively on the fungus. But I do not find, in my own observations, any material, either for the support or rejection of this theory, and I shall, therefore, refrain from discussing it. On the other hand, these results suggest to my mind that a most effectual and rapid cure would be brought about if, by any artificial means, a

complete shedding of the hair of the affected part be produced; as, in this way, the hair would be cast off from its very connection with the papilla, instead of being broken off at some higher part of its course, as in the process of epilation. Biesiadecki, in Stricker's 'Histology,'¹ thus describes the natural falling of the hair.

"The fall of the hair results from the circumstance that new cells form around the papilla. The last formed cells of the hair root, receiving no impetus from behind, become converted into hair-substance, and form either the conical or club-shaped inferior extremity of the shaft, which is composed of fibrillated hair-scales. * * * * The sheaths of the hair-follicle, which in consequence of their muscular structure (?) exert a constant pressure upon the contents of the hair-follicle, force the clubbed extremity of the hair, together with the sheath of Huxley, continuously upwards, and so contract that the vitreous membrane presses directly on the papilla; whilst at a higher point the internal surfaces of the external root-sheath come into contact. The new hair is developed from the old papilla."

If, then, the hair is affected with fungus, it will be seen that its separation from the papilla by the contraction of the follicle-wall would put a stop to further infection; the vital outer root-sheath acting throughout as an effectual barrier between the fungus-loaded hair in front, and the healthy young hair behind. Amongst my sections, I was fortunate in meeting with that of a follicle in which the hair is in the act of being shed, and I have made a drawing of it, as illustrating the point suggested (Pl. II, fig. 5). The very small portion of hair figured is healthy, and I am unable to say what was the condition of that in the upper part of the follicle; but it is clear that the new hair would be quite protected from infection by the old one. On the assumption that the fungus grows freely in the root-sheaths, and even in the papilla, as some seem to suppose, no cure could be expected from such a process; but the limitation of the

¹ 'Handbuch der Lehre von den Geweben,' vol. i, p. 612, and Syd. Soc. Translation, 1872, vol. ii, p. 257.



disease to the structure of the completely formed hair, which these observations so strongly demonstrate, is precisely the arrangement which renders possible its extermination in this way. How often a cure is brought about thus, must be still uncertain. It is at least possible that some forms of irritation, such as blistering, may act in this manner; but, with this exception, the irritations employed for ringworm are much less severe than that spontaneous inflammation of the skin, namely, erysipelas, which most often causes shedding of the hair. On the other hand, one would expect some falling of the healthy hairs also, if the irritants used in the cure of *Tinea* commonly acted as above suggested.

This at least seems to me worthy of consideration that could any means be employed to cause the hairs to be thrown off from the papilla, a cure would more certainly and more quickly result, than by the methods commonly in use.

In conclusion, I would suggest to future workers in this field, that much may possibly be learnt by observations on the skin in a stage of *Tinea tonsurans* advancing towards cure, a stage which, for the reasons explained, had not yet been reached in the present case.



DESCRIPTION OF PLATES I AND II.

On the Skin in *Tinea tonsurans* (Dr. F. Taylor).

Plate I, fig. 1.—Transverse section of a follicle with diseased hair. *a.* Outer root-sheath. *b.* Inner root-sheath. *c.* Spores of the fungus surrounding the hair. *d.* The hair-shaft split up by mycelium-threads and spores. Magnified 140 diameters.

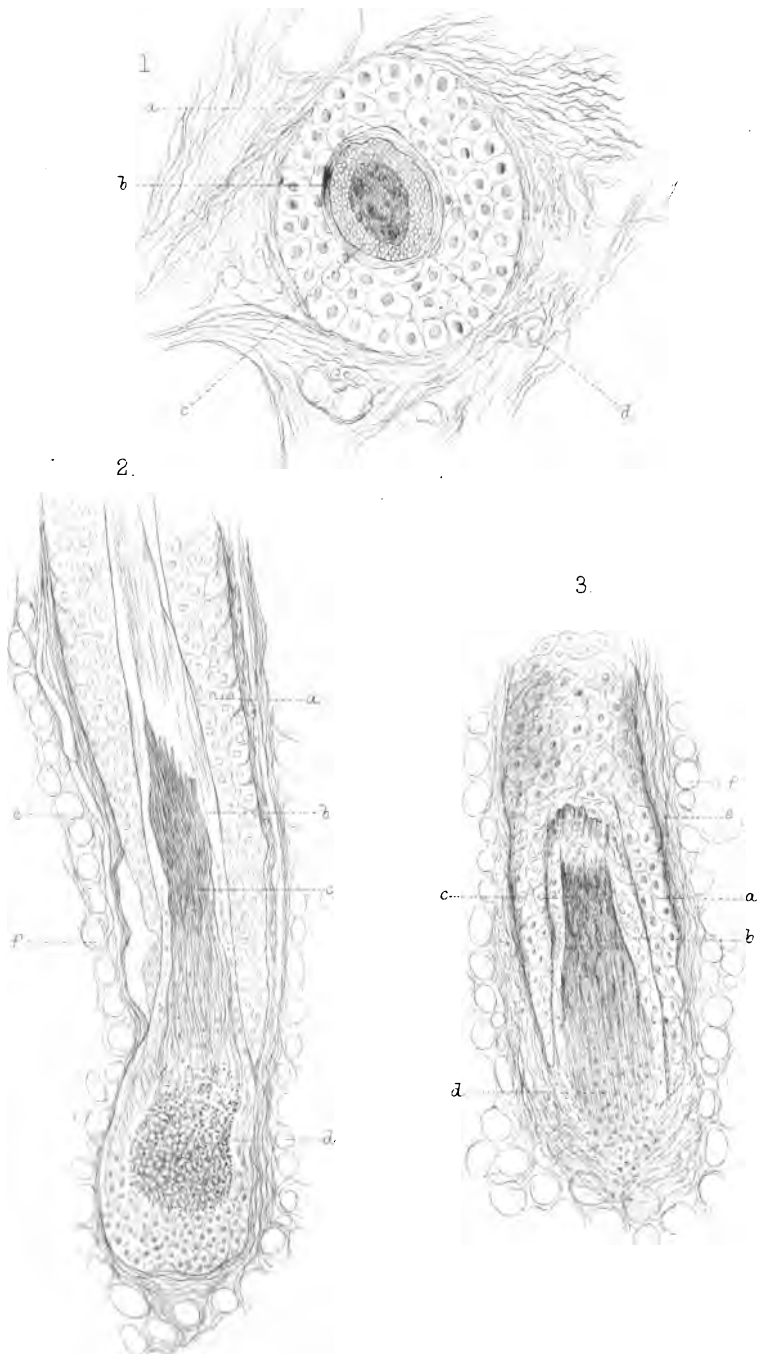
N.B.—In this drawing and in Fig. 4 the spores are represented about twice as large as they should be. According to the scale given their size would be about $\frac{1}{8000}$ inch; their actual measurement was under $\frac{1}{10000}$ inch.

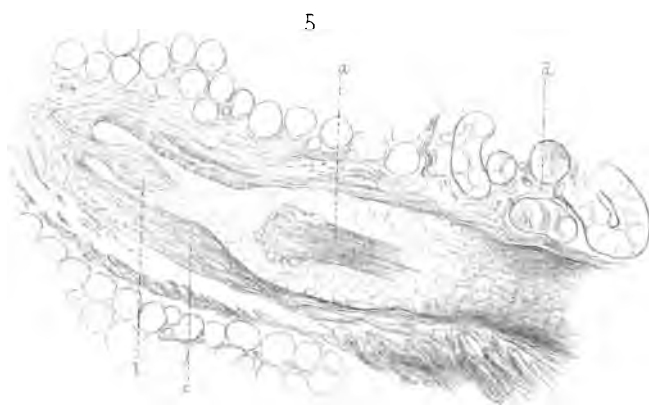
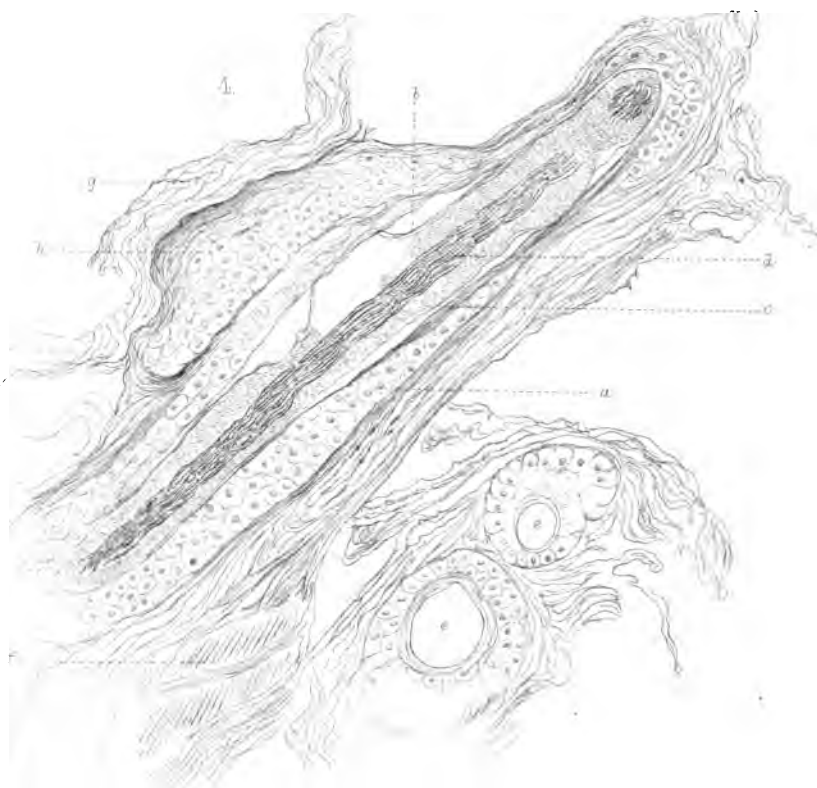
Figs. 2 and 3.—Longitudinal sections of hair-follicles. *a.* Outer root-sheath. *b.* Inner root-sheath. *c.* Hair filled with mycelium-threads. *d.* Soft pigmented cells surrounding the papilla. *e.* Wall of the follicle. *f.* Fat-cells. Magnified 104 diameters.

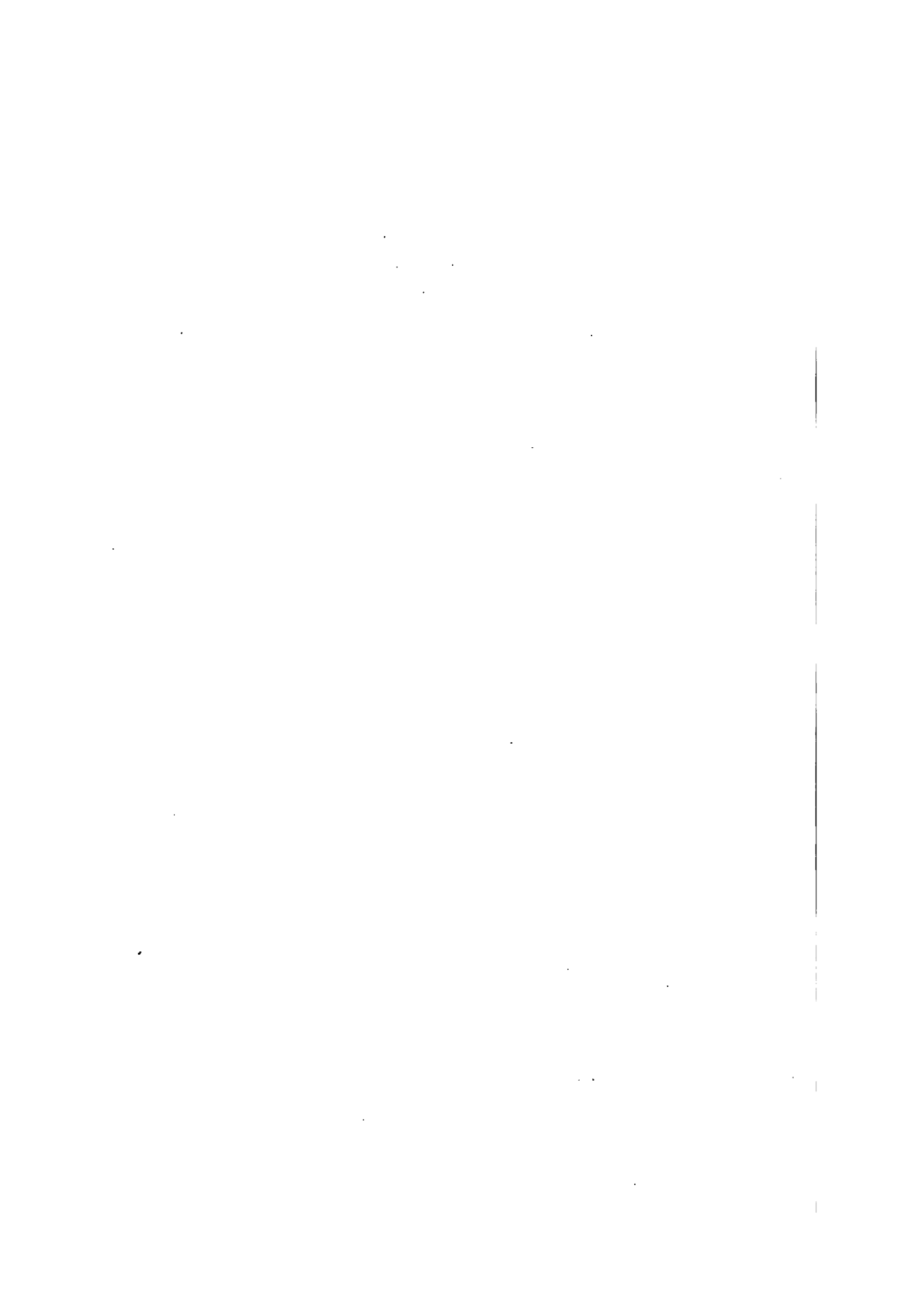
Plate II, fig. 4.—Longitudinal section of hair-follicle, running obliquely under the skin. *a.* Outer root-sheath. *b.* Inner root-sheath. *c.* Fungus-spores. *d.* Hair-shaft destroyed by mycelium and spores. *e, e.* Transverse sections of follicles from which the hair-sections have fallen out. *f.* Arrectores pili cut obliquely. *g.* Horny layer of the epidermis half detached by maceration. *h.* Mucous layer of the epidermis. Magnified 100 diameters.

Fig. 5.—Longitudinal section of follicle and papilla, showing the fall of a hair. *a.* The shaft of the hair, which terminates in altered cells, and does not form the usual bulb. *b.* Papilla. *c.* Walls of the follicle closely contracted upon the papilla (compare Fig. 2). *d.* Sudoriparous gland. Magnified 104 diameters.

1001







THE NATURE
OF
IODIDE-OF-POTASSIUM ERUPTION.

BY
GEORGE THIN, M.D.

(Received October 28th.—Read November 13th, 1878.)

It is now established that the administration of preparations of iodine usually produces an eruption which varies in character in different cases. All practitioners of experience are familiar with the papular eruption or common iodine-acne, but the combined testimony of a considerable number of observers during the last decade has shown that the rash sometimes assumes a more severe type. In such cases the iodine eruption may be pustular, or in the form of bullæ or blebs containing serous or sero-sanguinolent fluid, or purpuric, and more than one of these forms may be found together on the same patient.¹

¹ See (*e. g.*) Cazenave, quoted by Boinet in his work, 'Iodothérapie;' Bradbury, 'British Medical Journal,' 1871; Bumstead, 'American Journal of the Medical Sciences,' 1871; Ringer, 'Practitioner,' 1872; Report by the Registrar in the 'London Hospital Report,' 1875 (a case of Mr. Hutchinson's); Fournier, 'Revue mensuelle de Médecine et de Chirurgie,' 1877; Taylor, "Report of Meeting of the New York Dermatological Society," in the 'Archives of Dermatology,' 1877; Dr. Tilbury Fox, 'Transactions of the Clinical Society,' 1878; Sydenham Society's 'Atlas,' pl. xxxiii.

The explanation usually given of these eruptions, namely, that they are produced by stimulating effects on the secreting elements of the sebaceous glands, is, in the absence of direct proof that there is a special elective affinity on the part of these organs for iodine, only a fanciful one, and rests on no better foundation than the fact that the vascular tissues surrounding the hair-follicles and hair appendages are frequently the seat of congestion when the intermediate less vascular tissues of the cutis show no evidences of it.

I take the opportunity of submitting to the Society the results of a microscopic examination of a portion of skin which was the seat of a bullous iodide eruption. The patient was under the care of Mr. Howard Marsh in St. Bartholomew's Hospital, and it is through the kindness of this gentleman that I am able to report on the case. Mr. Marsh has also been so good as to furnish me with the following history.

"W. R—, æt. 35, was sent from the City Road Hospital for Diseases of the Chest, by Dr. Wharry, to St. Bartholomew's Hospital on May 18th last. In his letter Dr. Wharry stated that the patient, who had renal and mitral regurgitant disease, with hypertrophy of the heart, had been taking either iodide of potash or syrup of the iodide of iron, continuously for tertiary syphilis, from Feb. 1st to May 10th, when the medicine was discontinued. On May 13th the present eruption began. On his admission (May 18th) on the right cheek was a bulla (?) of irregular shape, about as large, at its base, as a shilling, rising abruptly and prominently from the surface, and apparently containing a turbid, opaque, cream-coloured material, of the consistence of thick pus. The base of this 'bulla' was somewhat solid and indurated, dusky red in colour, and very irregular in outline, as if it were formed by the coalescence of two or three smaller 'bullæ,' but this was uncertain; the surface was covered with a brownish scab or crust tending to dry. A very similar 'bulla' was situated on the right cheek just below the eye, and on the left side of the tip of the nose was a third, about as big as a sixpenny bit. Scattered over the face were several

-small 'pustules,' varying from the size of a pin's head to that of a large split pea; and one seated on the left ear remarkably resembled a pemphigus bleb. A small pustule was situated on the left wrist, and one, somewhat larger, on the dorsum of the right hand.

"The patient died suddenly on May 22nd."

The rash was considered to be due to iodide of potash by Mr. Marsh, Mr. Baker, Mr. Langton, and by Dr. Crocker, who saw the patient on May 20th.

The piece of skin examined was from the dorsum of the right hand.

The diagnosis, vouched for by Mr. Marsh, Mr. Baker, Mr. Langton, and Dr. Crocker, leaves little room for comment, but it may not be superfluous to state that Mr. Marsh's description of the eruption agrees in every respect with that given of iodide bullous eruptions by previous observers.

I received the portion of skin in chromic acid solution. A dark spot about the size of a pin head marked the centre of the bulla.

The tissue having been further hardened in alcohol, an examination of vertical sections showed the structure of the skin over which the bulla was situated to be as follows:

I find that the diseased tissue is divisible into three well-marked areas. In the centre there is a faintly granular, almost homogeneous, mass, within which here and there rows of leucocytes can be seen. This mass is mostly covered by a ragged and partly disintegrated epidermis. The substance of which it is composed is identical in appearance with a like substance often found in any part of the body when there has been effusion from blood-vessels in diseased tissues. In such cases it is sometimes found both within and outside the vessels. A layer of it enters into the composition of a scab. Its derivation from the blood is beyond question.

Proceeding from this structureless substance as a centre we next find a layer of exudation cells. The cells are suspended in a very fine reticulum which is probably fibrinous. Occasionally in this area the section of a blood-vessel is

observed, the calibre of the vessel being indicated by a thin, ragged wall, bounding a mass of red blood-corpuscles. Beyond this area comes a third, in which connective tissue can be recognised in a much altered condition. The bundles are represented only by fragments, and these fragments are separated from each other by spaces of considerable size. In the spaces are found exudation cells and a reticulum of fine fibres, similar to that found in the second area.

Amongst the disorganised bundles, sections of numerous blood-vessels filled with red corpuscles are seen, their walls showing more or less evidence of alteration.

This area is bounded by the normal bundles of the cutis tissue, the line of demarcation being very fairly defined.

The appearances I have described indicate effusion from the blood-vessels within a limited space, in sufficient force to produce disruption in one, and partially displace in another area the firmly knit bundles of the connective tissue of the skin. Such pressure from effused blood fluid in a limited area can only be produced by disorganisation of the walls of blood-vessels, and, in fact, vessels with altered walls are found in both the second and third areas of my division.

Appearances found in the unaffected connective tissue surrounding the bulla area, throw further light on the subject. Amidst the perfectly unaltered bundles blood-vessels are found, which in a limited and well-defined part of their course are plugged and distended by a faintly granular substance, similar to that found in the large mass in the central bulla area. At this part the distended vascular wall may be entire in so far as it is seen, or evidence of change may be detected in it. A considerable number of exudation cells are always found immediately outside the vessels, and sometimes the vessel appears slung in a space, across which, from the vessel to the connective tissue, fine fibres can be traced. There are exudation cells present in this space between the vessel and surrounding connective tissue. There was no evidence of disease in the hair follicles

or sebaceous glands immediately external to the disorganised tissue, and there were no remnants of epithelial or sebaceous elements found within it.

These appearances are sufficient to explain the formation of the bulla. They show evidences of a localised affection of the blood-vessels in a circumscribed area of the skin, attended by effusion of constituent parts of the blood. Part of this effused material is found coagulated after treatment by chromic acid and alcohol, and is evidently richly albuminous. The pressure is sufficient to displace the bundles of connective tissue and to rupture the mucous layer of the epidermis. The more fluid element of the blood necessarily escapes through the ruptured rete mucosum, and accumulating under the horny layer, as is the case with all similar effusions, a "bulla" is formed.

The size of the bulla and the nature of its contents will depend in such cases on the degree and extent to which the vascular wall is damaged, and on the size of the affected vessel.

That this injury to the blood vessels is independent of the glandular or epidermic elements of the cutis is shown by parts of blood vessels which are not in contact with, or even in immediate proximity to such elements, being affected.

The rationale of these eruptions, therefore, seems to be that there are conditions in which iodine, when present in the blood, attacks and disorganises the blood vessels at certain localised points. As a result of this injury to the wall of the vessel, there is an escape of blood fluid into the surrounding tissue, and more or less plugging of the vascular tube by coagula. It is a subject well deserving further inquiry, whether and to what extent the blood vessels of other organs are similarly affected.

The demonstration of an affection of the blood-vessels as a cause of iodine-bullæ suggests an explanation of the other changes known to be produced in the skin by iodine; namely, the common papular eruption and the rare, but now well-authenticated, instances of iodic purpura.

The papule of iodine-acne consists of a circumscribed area

of infiltration which usually surrounds a hair follicle. The redness and swelling of this area considered in the light of the acquisitions of modern pathology, are evidences of a localised affection of the blood-vessels, the moderate amount of exudation indicating that the affection is very limited in extent and degree. The tendency of iodine to exert its influence in a limited area, as illustrated by Mr. Marsh's case, may explain the formation of the iodic papule.

Iodic purpura can be looked on as the result of complete destruction of part of the vascular wall, with the effusion of a certain quantity of blood and the definite and sudden arrest of the circulation through the ruptured vessel—a circumstance that probably explains the absence of œdema. Probably the morbid change is here again strictly localised and it can only be produced by some destructive effect on a limited part of the vessel.

It may thus be considered that the iodic papule, the iodic bulla, and the iodic purpura spot represent different degrees of injury to the blood-vessels: in the first we have a limited œdema with congestion of the vessels; in the second an effusion of serum with more or less of the formed elements of the blood; in the third destruction of the wall of the vessel and hæmorrhage. The strictly local and limited action of iodine which I have found to produce the second, explains the localisation in the first and third.

In certain persons and in certain constitutional idiosyncrasies, the effects of poisons are unusually severe, and the action of iodine is in this respect not exceptional. The explanation of these rarer and more severe symptoms can only be guessed at, but it is not unreasonable to suppose that long retention of iodine in the capillaries when the circulation is sluggish from feeble heart-impulse, its deficient excretion from a similar or other cause, and a diminished vitality of the vascular wall which renders it more susceptible of attack, may each or all of them play an important part in the work of destruction.

It is only on some such hypothesis that a remarkable

feature in this case can be accounted for. The patient had been taking preparations of iodine from February 1st to May 10th without any eruption showing itself, and he then ceased to take the medicine. Three days after he had discontinued the iodide the eruption began, and on the ninth day afterwards he died.

The history of the case shows pretty clearly that the loss of vital power which ended in death must have been in operation before the last iodine taken into the system had been eliminated, and it is plausible to assume that the circulation becoming slow in the superficial vessels the iodine or its compounds present in the blood had time to attack and injure parts of the vascular wall.

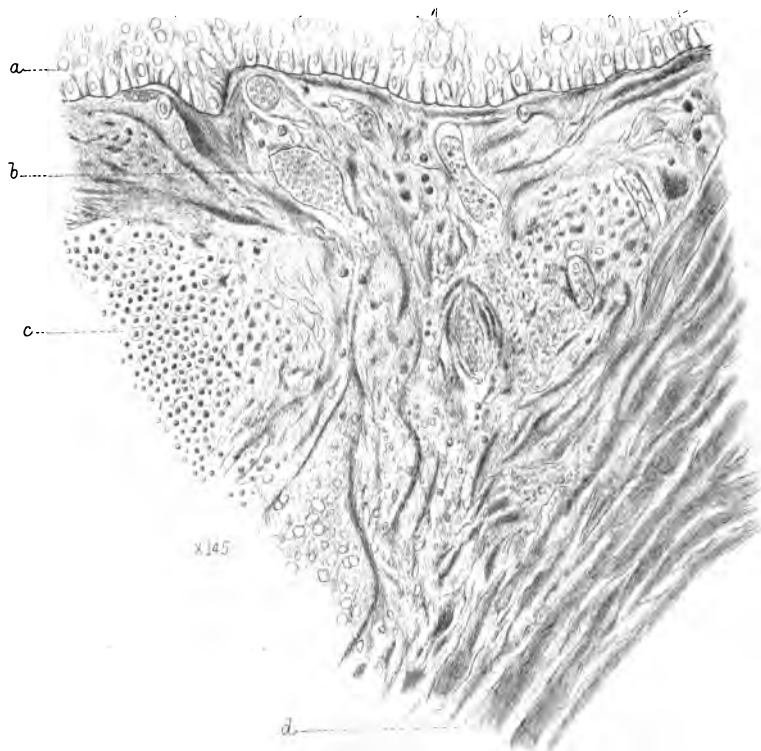
DESCRIPTION OF PLATE III.

Iodide of Potassium Eruption (Dr. G. Thin).

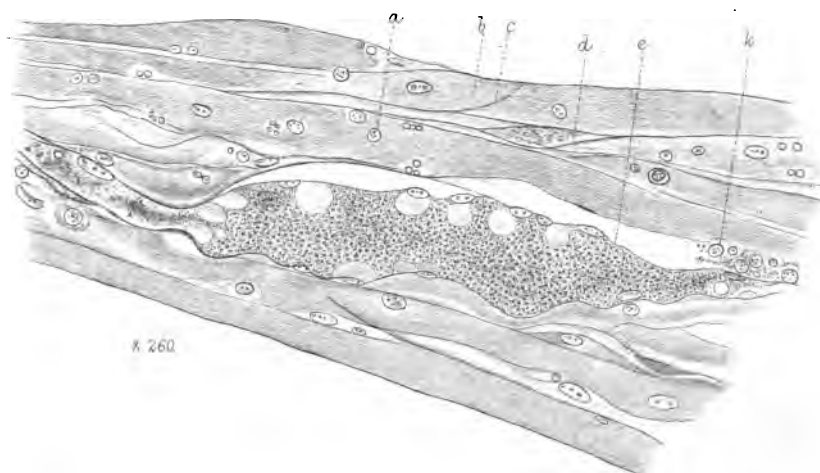
Fig. 1.—Part of a section showing the second and third areas described in the text. On the left border towards its centre the cell reticulum is seen, and beyond that, narrowing on the right towards the rete mucosum is the third area of disorganised connective tissue, bounded below and on the right by normal bundles. *a.* The rete mucosum. *b.* Section of a blood-vessel. *c.* Mass of exudation cells lying in a delicate reticulum. *d.* Normal connective tissue. $\times 145$.

Fig. 2.—Shows part of a blood-vessel distended and plugged by granular coagulated substance. The vessel is surrounded by normal connective-tissue bundles. *a.* Exudation cells. *b.* Normal bundles of the connective tissue of the cutis. *c, d.* Elastic fibres. *e.* Distended and plugged vessel. (On the border of the vessel nuclei of the vascular wall are seen. Retraction during coagulation or hardening by chromic acid has left rounded spaces on the surface of the mass). *k.* Exudation cells between the vessel and the contiguous bundle. $\times 260$.

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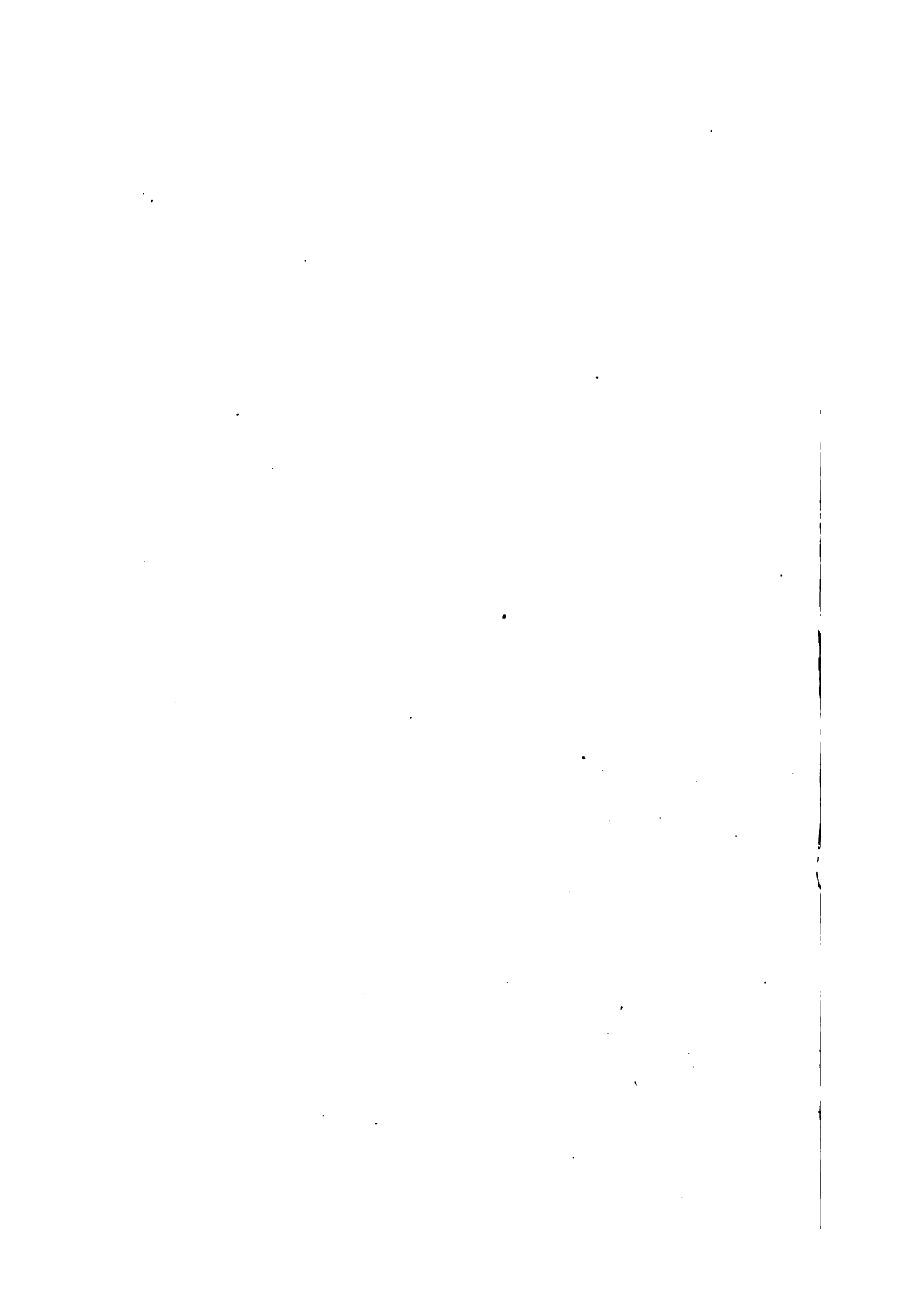


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TRACHEOTOMY IN MEMBRANOUS LARYNGITIS:

THE INDICATIONS FOR ITS ADOPTION, AND SOME
SPECIAL POINTS AS REGARDS ITS
AFTER-TREATMENT.

BY

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(Received October 22nd—Read November 26th, 1878.)

“Un remède expérimenté vaut mieux qu'un désespoir assuré.”

TRACHEOTOMY is performed for various diseases involving the larynx, either directly or indirectly. It is, however, with cases of membranous inflammation of the larynx and trachea, either with or without pharyngeal complication, that I am at présent concerned. It will not be necessary to discuss the identity or otherwise of these diseases, as the principles of surgical treatment, which I venture to submit for consideration will, I believe, be found applicable to all cases where laryngitis is accompanied by a deposit of membrane, leading to mechanical obstruction, whether they be called croup or diphtheria.

Before entering on the indications for the performance of this operation, I hope I may be pardoned, if I venture to

express regret that the surgeon is only too often called in after all therapeutic measures have failed, and the more so because these measures have generally included the use of depressants, which if not at once beneficial, have by their continued administration tended greatly to increase prostration, so often a predominant feature of the disease.

Briefly put, one may say, that tracheotomy is indicated in cases of increasing or persistent dyspnoea, due to mechanical obstruction in the larynx or trachea; the best test of the degree of this mechanical obstruction not being the apparent distress of the patient, but rather the amount of the recession of the chest wall, and supraclavicular spaces, especially in conjunction with suppression of voice, more or less complete.

The loud clanging cough, which is such a source of terror to the anxious parent, is really far less ominous than the hoarse whisper and the almost inaudible cough, which mean direct implication—one might almost say, with Niemeyer, inflammatory infiltration and paralysis—of the narrow chink of the glottis.

The indications for tracheotomy are the more urgent, when expiration, as well as inspiration, are alike laboured; for neither spasm nor paralysis will explain impeded expiration.

To wait for distension of the jugular veins, and until general cyanosis have supervened, is postponing the operation beyond the limits of mere mechanical obstruction; it is waiting for the advent of pronounced carbonic acid poisoning.

After deciding upon the operation, the first point, which has to be settled is, shall the patient be chloroformed? I have never seen any harm result from this proceeding; a very little chloroform usually suffices, and therefore advantage ought to be taken of the facilities, which it undoubtedly offers for a careful and deliberate operation. Care and judgment must of course be exercised in each individual case.

Coming to the operation itself, I agree with those authors who advise the high operation. In young children, the high

is more easily performed than the low operation. It would often be more correctly named crico-tracheotomy, involving, as it probably nearly always does, the cricoid cartilage and one or more tracheal rings. I have never seen any inconvenience result from cutting the cricoid. The position of the isthmus of the thyroid gland varies considerably in children. I have more than once seen it lying across the cricoid cartilage; it is, however, more frequently situated over the upper two or three tracheal rings. Whenever the isthmus is high, it is better to incise the cricoid, more space is obtained for the easy introduction of the tube; the old fear of its non-uniting or necrosing is, in my experience groundless. Should the isthmus ever be found really in the way it may be divided without hesitation, though it is perhaps better to depress it with the handle of the scalpel or with a blunt hook, whenever this is possible.

In common with Mr. Holmes and Mr. Marsh, I would insist on the advantage of thoroughly exposing the trachea before it is incised, for this allows us to make our opening with precision, and moreover facilitates the introduction of the canula. I believe also the danger of infiltration of pus into the muscles and intra-muscular planes during the subsequent course of the disease is diminished.

One of the greatest and most frequent impediments to a rapid operation is hæmorrhage. The veins of the neck are very numerous, and being over-distended they bleed freely when incised. It is difficult sometimes to know what to do, under these circumstances. I believe, however, that I may venture to advise what not to do; do not tie them, for this may give rise to thrombosis, which may prove dangerous from extension into the large veins. The hæmorrhage does not continue as a rule; if it be severe a pair of Dieffenbach forceps may be temporarily hung on; they can be removed as soon as the trachea has been opened and respiration re-established. I have never known hæmorrhage continue, or recur after the operation was once completed.

I would suggest the methodical use of a dilator after the trachea has been incised, rather than the immediate intro-

duction of the canula, because shreds of membrane and thick muco-pus can be expelled through a slit in the trachea more easily than through a silver tube.¹

I advise that a large feather be passed into the wound, downwards into the trachea, and upwards towards the glottis, so as to detach all the membrane as completely as possible. The presence of membrane or inspissated mucus in the larynx, above the tube, after tracheotomy, is often an unsuspected source and cause of reflex cough and irritation; there is a not unnatural tendency to the accumulation of exudation products in the larynx; some of them may have been deposited before the operation, a portion may possibly collect afterwards. In either case, the surgeon ought, every now and then, to clear out the larynx, so long as the patient is unable to do this for himself by natural means; and while he has to wear the canula in his trachea, he is unable to use the natural means, viz. coughing, owing to the fact that all air is diverted, from the larynx, through the tube.

This clearing away of the local manifestations of the disease is of the first importance, and it should be done in all cases before the child is removed from the operating table, seeing that it can be done more easily before than after the insertion of a tube, provided a suitable dilator be used. I venture to surmise that, after many a well-performed operation, in which, notwithstanding a careful incision of the trachea, the insertion of the tube has not been followed by relief, the secret is to be found in the fact that the trachea has not been cleared out. The tube may actually pass between the tracheal wall and a complete membranous cast,

¹ Mr. Holmes ('Surgical Treatment of Children's Diseases,' page 322) says, "If the air-tube has been well exposed and freely opened, a great gush of air instantly comes out, accompanied by violent spasmodic cough, and the free expectoration of blood, mucus, and false membrane, if present;" and in a footnote he adds, "False membranes may *sometimes* be seen in the wound. In such cases it is well to draw them out, and to wait for some little time before putting in the tube." It will be seen that I go further than this author, and would urge the importance of thoroughly clearing the trachea and glottis of all foreign matter in *every* case as a matter of routine, whether shreds of membrane protrude or not, before the introduction of a tube.

which it has but partially thrust aside. Indeed, I have a specimen illustrating this. The too-anxious surgeon often feels that his patient is not safe until the tube is inserted and he hurries to accomplish this. But let it be remembered that, with a dilator in use, immediate danger need not be apprehended; while, on the other hand, a tube inserted too soon may prove but a false security.

The trachea and larynx having been cleared out by means of a feather the tube may be inserted. Contrary to the advice of some English authorities,¹ I would advise the use of the largest tube which can be got into the trachea, without actual violence.

These surgeons argue that there is no need "for a larger opening than nature gives us." But I do not think that the narrow chink of the glottis ought to be compared with a silver tracheotomy tube. The depth of the former at the most is one quarter of an inch, the length of the latter at least one inch. Even supposing other things were equal, the mechanical power required to draw in air, or expel air and exudations, would vary in proportion to mere length or depth of the tube—the resistance or friction increasing, of course, with the length. This can easily be appreciated by any one who will endeavour to breathe, for some little time, through tracheotomy tubes of different lengths and sizes. Then again we have to remember that the strength of the patient is below normal, while the call for air—that is, the required number of respirations per minute—is increased in consequence of the disease, and possibly because a portion of the lung being *hors de combat*, the rest has to do duty for the whole.

Another very important advantage in using a large canula is the comparative facility with which the tracheal secretions are got rid of.

For these reasons, I venture to recommend the largest-sized tubes that can be got into the windpipe without actual violence; further, I would advise that they be as short as is consistent with safety. Seeing that scarcely any part of the

¹ Marsh, 'St. Barth. Hosp. Rep.,' vol. iii, p. 341; Holmes, *op. cit.*, p. 324.

body is liable to vary in size and shape more than the neck, I refrain from giving any precise measurements for the tubes, but would say that the particular curve, calibre, and length of tube, will vary with each individual case, and can only be decided at the time of the operation.

The most useful canula that I am acquainted with, is the one which M. Roger, I think, originally devised (Fig. 1). Its essential feature is the moveable collar.

FIG. 1.



Drawing of tube, showing mechanism of the moveable collar.

FIG. 2.



Profile drawing. The dotted outline shows the degree of movement

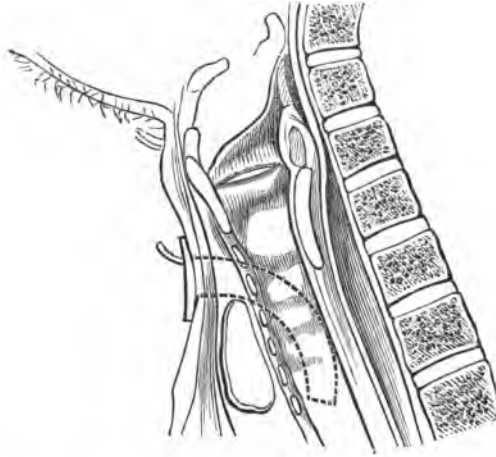
Fig. 2 shows how the tracheal part of this instrument can alter its position, and hence its curve, and how therefore it can adapt itself to a variety of necks, and to the ever-varying movements, which the trachea undergoes in talking, breathing, coughing, swallowing, &c.

The curve of the canula is a matter of great importance. For the most part, tubes are made in quarter circles. I do not consider the quarter-circle to be a good curve to adopt; in my opinion, it is from this circumstance *chiefly*, that ulceration of the anterior wall of the trachea is now and then met with. This is not remarkable when we consider the direction which the trachea takes.

The outline figure is copied from Braune's 'Atlas of Topographical Anatomy,' and its correctness was tested by a preparation which I made this summer at University College, with the aid of my friends, Mr. Godlee and Mr. Ottley. The

tube, in dotted outline, represents an ordinary canula of the usual quarter-circle curve. I think the drawing fairly demonstrates that such a tube must almost necessarily impinge and press upon the anterior wall of the trachea.

FIG. 3.



Sketch of trachea, with a dotted outline of the ordinary tube *in situ*.

With a view to diminish this risk, I now propose a differently shaped canula (Fig. 4). The direction of this tube,

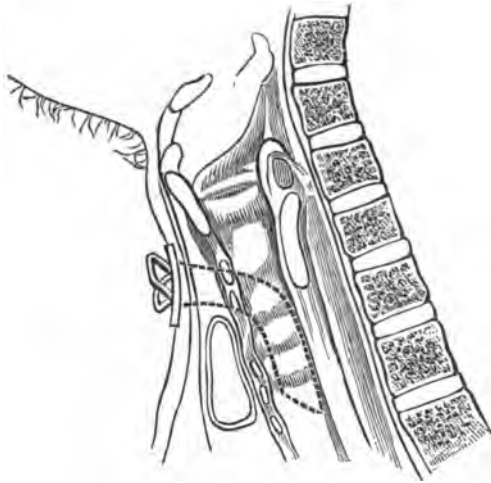
FIG. 4.



downwards and backwards, corresponds with that of the trachea. Such a tube can hardly press upon the anterior

wall of the trachea, and its moveable collar will also prevent pressure at the posterior part. I venture to think that it will be found useful. Fig. 5 shows the tube *in situ*, the outline figure being the same as that used in Fig. 3.

FIG. 5.



Sketch of trachea, with a dotted outline of the new tube *in situ*.

In describing the curve of such a tube, one may say, that its outline should approximate to the *Gothic*, rather than to the *Roman* arch. The tubes should, in all cases, be as short as is consistent with safety, and the lower orifice should be made at an acute angle, rather than at a right angle to the long axis, for this again lessens the danger of friction against the anterior wall of the trachea, and it also increases the area of the opening.

Mr. Baker,¹ believing that many disadvantages and some dangers attend the use of rigid tubes, has invented a flexible tube.

While I very much admire the ingenuity of the idea, and the excellence *per se* of flexible tubes, I cannot quite

¹ *Vide* the Society's 'Transactions,' vol. lx, p. 71.

subscribe to the imputations cast on the silver (rigid) tubes. Mr Baker would seem to imply that the dangers are due to the mere fact of the tubes being rigid, and no allowance seems to be made, either for a possible want of special knowledge and experience in the treatment of these cases, or for the use of ill-fitting tubes. When we consider the circumstances under which tracheotomy is so often performed, and that the operation is done chiefly by those who have had little or no experience, either in its performance, or in its after treatment, and further, when we come to think of the very small number of tubes which the surgeon, for the most part, has at his disposal, it will be conceded I believe, that there are other and potent reasons in explanation of some of the troubles which develop during the after-treatment of tracheotomy.

Ulceration is, in many cases, no doubt, produced by the pressure of an ill-fitting tube, but, under similar conditions, I believe, that the flexible tube would also produce a like result. I cannot think that pressure, sufficient to alter the curve of a flexible tube, could be exerted by the trachea, without giving rise to ulceration.

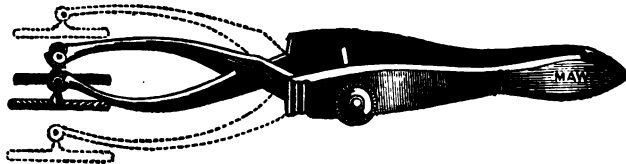
In other cases the granulations and ulceration are simply the result of the presence of a foreign body—to all intents and purposes, a canula is such—in the trachea, and in such cases the rubber would be just as likely to set it up as the silver; much on the same principle that a foreign body on the eye gives rise to inflammation, whether that foreign body be a fly, a grain of sand, or an inverted eyelash.

Cases of tracheal ulceration have occurred in my own practice, and that too, although I was fully alive to the possibility of its occurrence, and though I endeavoured to avoid it. I do not attribute this to mere rigidity of tube, but rather to my own want of skill, and to the use of tubes not suitable to my case. While, therefore, I most willingly admit the utility of flexible tubes, I nevertheless, for the present, reserve my judgment as to their comparative value. We must wait until their more extended use enable us to look back on the results obtained in other hands than those

of the inventor, before we can offer any reliable opinion on this point.

While on the subject of instruments, I would just allude to a little "automatic retractor" which I have had made. I devised it after having been once or twice suddenly called on to perform tracheotomy when there was no one to assist me, and I need scarcely say that it is a very awkward operation to do alone. Under such circumstances the "automatic retractor" will be found useful. The incisions into the soft parts having been made, the blades of the retractor are introduced closed (like an eye speculum), they are then opened to the required extent, and the screw is adjusted.

FIG. 6.



Automatic retractor.

It does not take the place of a good assistant, but it may occasionally be found valuable, when an assistant is not at hand, not only in tracheotomy, but even also in hernia, in the removal of small tumours, and in other and like operations.

Passing on to the after-treatment, properly so called, I ought first to mention, as perhaps, the most important, the advantage, indeed, the absolute need of well-trained nurses to take charge of the cases after the operation.

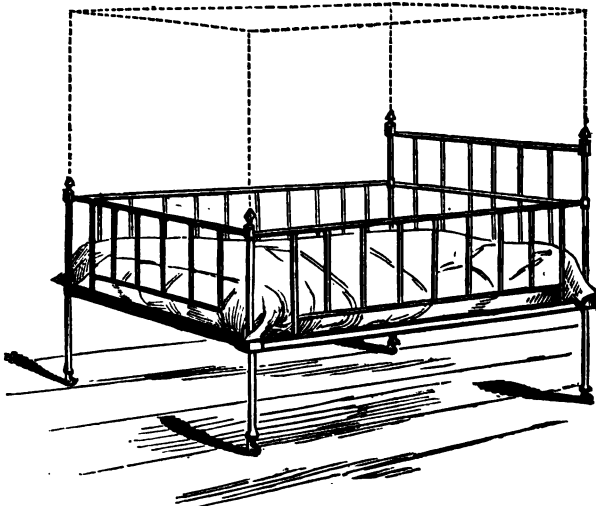
If we constantly keep in mind the chief object of the operation, viz. the easier admission of air into the lungs, we get one of the most important and useful indications for treatment. This indication I would formulate as follows:—*The air-tube is to be frequently and carefully cleared, in some way or other, of any retained membrane or other secretion, as soon as its presence is suspected. Its presence may be suspected among other symptoms, 1st, by progressive dyspnoea,*

general malaise, and restlessness ; 2nd, by a whistling sound heard with each breath ; 3rd, by suppression of the tracheal secretion which the presence of a canula ought itself to set up.

The presence of membrane in the trachea is dangerous from a two-fold point of view. 1st, it is dangerous because it mechanically prevents the entrance of air into the lungs ; and 2nd, because it is infective. I would therefore advocate its early and complete removal and destruction, just as I should endeavour to get rid of it if I saw it attack an external wound.

Various means may be used to facilitate its removal. By far the most important of these is the inhalation of steam. For this purpose, the patient's bed should be surrounded with curtains. The plan I found in use at the Hospital for Sick Children, seems to me the most practical and best adapted to its purpose. A rod (wood or iron) is strapped to each one of the four legs or supports of an ordinary child's

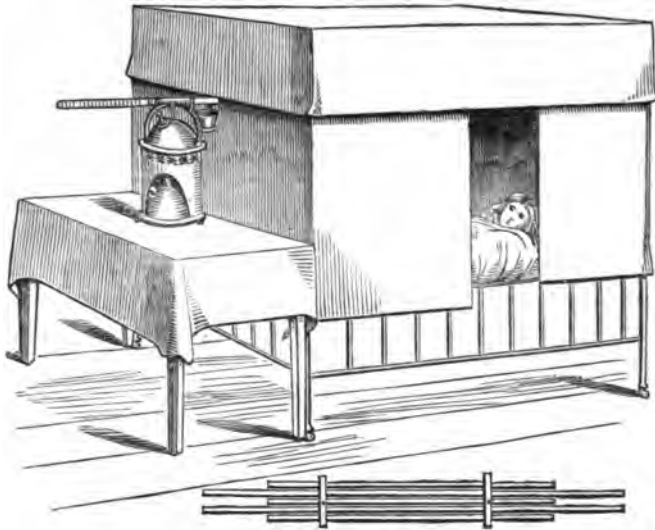
FIG. 7.



An ordinary cot. The dotted lines show the iron framework.

cot; these four uprights are then connected together by four other cross rods; in this way an open cot is converted into an old-fashioned four-post bed. Sheets are then thrown over this extemporised framework so as to cover the top, the two ends, and one side. The diagram explains the plan better than any words can.

FIG. 8.



The croup-bed complete.

The plan, at use in some hospitals, of drawing the bed close to the fire-place, surrounding it with the ordinary ward screens, and then covering them in with blankets, does not seem to me so good a plan as the one I have just recommended; for it is more difficult to regulate the temperature, and it frequently deprives the patient both of the fresh air and light which are so necessary for his recovery.

A *small* steam apparatus is kept boiling at the foot of the bed, and the requisite amount of steam is conducted inside the curtains.

The amount of steam required varies with the individual case. *An excess, however, is in all cases to be avoided, as it*

tends to depress the patient. The indication for steam, may, I think, be formulated thus:—The less there is of tracheal secretion, the more is steam needed; and the converse. Steam acts on the bronchial mucous membrane much as it acts on the skin; it encourages secretion; the secretion tends to loosen the membranous exudation, and so bring it within the current of the expired air. It thus facilitates its removal.

Steam, too, may be made the vehicle for important medication; thus, disinfectants, stimulants, sedatives or expectorants, may be added according to circumstances. The importance of systematically using disinfectants—especially in children's hospital practice—cannot be over-estimated. For this purpose either carbolic acid or kreasote may be used. The latter is especially valuable, since it possesses a decidedly stimulating as well as disinfecting influence. I have found it useful in cases where, owing to scanty tracheal secretion, the removal of membrane has been attended with difficulty. Amongst sedatives, the compound tincture of benzoin, with or without a few drops of chloroform, has given good results.

I shall now speak of *solvents*. By solvents I mean drugs, the action of which is to liquefy the membranous exudations. Among the most important are the alkalies, lime, soda, and potash. If portions of well-formed membrane be placed in different test tubes, severally containing a solution of saccharated lime, of carbonate of soda, of carbonate of potash, and of distilled water, very marked differences in result will be observed; the lime solution will dissolve the membrane in an hour or two; the soda and potash in a somewhat longer time; while the distilled water will not produce any effect for several days, and then only by decomposition. In any given case, where it is desired to liquefy the membranous exudations, one or other of these solutions may be tried. I myself prefer the soda solution. The lime is so quickly altered and rendered inert by the carbonic acid of the expired air, as to become practically useless. A solution of carbonate of soda (10—20 grains to an ounce of distilled

water) is very effectual. It may be sprayed in front of the canula and the spray inhaled; it is thus brought into immediate contact with the surface of the tracheal mucous membrane. This is to be continued for five or ten minutes, at longer or shorter intervals, according to circumstances. I do not wish to imply that this momentary contact of the soda with the membrane, acts in the trachea as it does after a lengthened interval in the test tube; fortunately, the membrane need not exist as such after the trachea has once been opened; for membrane is a gradual formation, and results, no doubt, from an aggregation of smaller particles. It is exactly on these smaller particles that the soda solution can, and I believe, does act, in the sense of liquefying them, of helping to detach them, and of rendering them less organisable.

Steam, often without any mechanical aid, materially helps in the expulsion of membranes; it is not well to trust to this alone. I make free use of large feathers,¹ and twirl them about in the trachea, so as not only to detach the membrane, but also to entangle it and draw it out through the tracheal wound. If a feather does not suffice, then recourse may be had to aspiration. For this purpose, a soft or elastic catheter answers very well. Care must be had to close the tracheal wound before suction is made, or otherwise, the aspiration is unavailing. The catheter may be inserted as far as it will

¹ The late Professor Trousseau at one time advocated mopping out the trachea. He says ('Clinique Médicale,' vol. i, Paris, 1861):—"When I first practised tracheotomy, following Brettonneau's example, I used to order the trachea to be mopped out, as low down as possible, with a sponge fixed on to a bit of whalebone. I have long discontinued this proceeding, as also cauterisation of the trachea which I used to practise. . . ." It is difficult to gather from this statement whether Trousseau's proceeding is comparable with my suggestion as to the methodical treatment of the trachea, either at the time of the operation or subsequently. It is, however, very probable that this treatment by Trousseau first gave me the idea of trying it. Trousseau, however (op. cit., p. 424), says, "It is a remarkable fact that, when once tracheotomy has been performed, we need not further occupy ourselves with the pharyngeal or the laryngeal manifestations of the diphtheria, which claimed such energetic treatment previous to the operation. They get well of themselves."

reach, without any fear, in cases where the disease has spread from the trachea into the bronchi. By this means membrane may be followed down deep into the chest, and success obtained, where without such means death would be inevitable.¹

The next question is, how often must the tube be cleaned, and how often must it be taken out. This will vary with each individual case. The interior tube may require changing every hour, or even oftener; when the temperature is high, the tubes also become heated, and the discharges the more quickly inspissate and adhere to the tube. The soda solution will be found useful in cleansing them.

Speaking generally, I would say that a free secretion from the trachea is not an unfavorable sign; on the other hand, a "dry tracheotomy" ought to be regarded with anxiety. These "dry" cases are especially tolerant of interference. I have seen a large feather introduced for several inches into the trachea, without giving rise to any reflex action. Of such cases I am always very suspicious, and should always give a most guarded prognosis.

The exterior tube, after the lapse of from twenty-four to thirty-six hours, ought also to be taken out and cleaned. This is an useful proceeding for many reasons; for it allows us to detect the earliest signs of unhealthiness in the exter-

¹ I may, perhaps, be allowed to say that much of this treatment will be found adapted to, and efficacious in, the early stages of membranous laryngitis. Indeed, I would recommend the use of steam with or without medicaments, and the spraying of the mouth and fauces with soda solution as soon as ever symptoms of laryngeal affection come on. If the disease commence primarily or at all severely in the pharynx, then the application of hydrochloric acid, as recommended by Bretonneau and Trousseau, is of great service. Strong hydrochloric acid, diluted with two or three times its bulk of glycerine, is the solution I would recommend. The secretion from a diphtheritic surface is peculiarly acrid and irritating; it tends by excoriating the mucous surfaces to spread the disease; it is, therefore, a source of danger, and it ought to be thoroughly destroyed. Whatever be the *modus operandi*—whether it merely substitute a simple for a specific inflammation as Trousseau taught, or whether it destroy the microphytes, which Oertel, among others, regarded as the source of the infection—matters little; it is beyond all question a most efficacious application.

nal wound, and to apply appropriate remedies. Occasionally too, the tube is found to be discoloured; this suggests decomposition, and warns us to look out for possible complications. I think it is a good plan to have two tubes, identical in everything except length, and to use them alternately; this is one means of anticipating and preventing erosions. There is not much difficulty, as a rule, in reintroducing the tube after the first forty-eight hours have elapsed; but it is safe to have a dilator at hand in case of need.

Another advantage of taking out the exterior tube is, the opportunity, thus afforded, of testing the breathing power through the larynx. It is well to commence these trials early—say about the third day—before disuse has crippled the muscles which open the glottis; the attempts may be renewed once or twice daily, according to the encouragement which any individual trial affords.

The final removal of the tube is always an anxious part of the after-treatment. In many cases it is accomplished without any difficulty, in a few cases, it is a source of great trouble and not a little danger. It would be outside the limits of the present paper to enter at length on this subject. I must content myself with the briefest allusion. It seems to me, the most troublesome cases are those in which the difficulties arise from inflammatory thickening of the vocal chords, and of the muscles which open them. This leads to what is practically paralysis. In such cases the patients can breathe for an hour or two, and then difficulty comes on; in others, the patients get on well enough as long as they are awake; but the moment they fall asleep—and when, therefore, the accessory muscles of inspiration are also asleep—their difficulties at once come on. It is important to persevere with such cases. The tubes must be removed for as long as possible every day, in order to reimpose on the larynx its normal functions. Then, as soon as difficulty arises, the tubes must be replaced, in order to give the inspiratory muscles rest. The reintroduction of the tubes after a few hours' absence is not always easy, on account of contraction taking place in the wound. Under these circumstances, I have

found it useful to insert a little silver plug just long enough to reach the trachea, without, however, doing more than keeping patent the wound. A piece of moulded gutta

FIG. 9.



Plug.

percha will answer the purpose equally well, if a silver plug cannot be had. On removing the plug, the tube can be easily inserted; if there is still any difficulty, then the dilator may be used. Each case must be treated on its own peculiar merits. Mr. Thomas Smith contributed an exhaustive paper on the subject to the Royal Medical and Chirurgical Society in June, 1865,¹ to which the reader is referred for further details on this important matter

Most surgical authorities agree as to the non-dangerous nature of the operation *per se*, and a careful study of many published fatal cases, inclines me to think that exceedingly few die from the effects of the operation itself. The most frequent cause of death is the supervention of pneumonia, This complication has been attributed to the opening of the trachea; but it is worth noting that pneumonia seldom ensues on operations done for other morbid conditions (e. g. warty growths, foreign bodies, &c.); whereas, pneumonia is *constantly* found post mortem in cases of diphtheria, which have *not* been tracheotomised.

* * * *

Bearing in mind then, that the operation is undertaken, not as a curative measure but simply with a view to relieve a mechanical impediment to respiration; seeing, nevertheless, the great frequency with which, after tracheotomy, the

¹ *Vide* Society's 'Transactions,' vol. xlviii, 1865, p. 227.

trachea and larynx, on the post-mortem table, are found covered, not to say choked up, with membranous exudation (specimens of which may be found in almost every anatomical museum), I think, as a practical outcome of the foregoing paper that I may venture to enunciate the following dictum.

The presence of membrane in the trachea in a fatal case of membranous laryngitis after tracheotomy, must be regarded as evidence of the want of due care on the part of the surgeon in charge, just as much as would the presence of a piece of gut in the inguinal canal after herniotomy, or a calculus in the bladder after the operation of lithotomy.

APPENDIX OF CASES, in which the after-treatment, as advocated in the foregoing paper, was carried out by the author, or under his supervision.

Total Cases, 21: recoveries, 12; deaths, 9.

Where operated on.	Physician.	Date.	Sex.	Age.	Recoveries.	Fatal.	Remarks.
<i>Hospital for Sick Children.</i>							
Dr. West	Feb. 20, 1873	M.	3 years	1	...	Pharyngeal as well as laryngeal; severe case; tracheotomy; inflammatory oedema of neck; no sequelæ.
Dr. Dickinson . .	.	June 2, 1873	F.	1 year 8 months	...	1	Had been ill one week; pharyngeal as well as laryngeal; antimonial treatment at first; was almost moribund when operated on; died on the third day.
Dr. Dickinson . .	.	Feb. 25, 1874	F.	3 years	...	1	Antimony at first, with temporary amelioration; died forty-eight hours after operation.
Dr. West	May 23, 1874	M.	2½ "	1	...	The boy developed scarlet fever the day after the operation; he nevertheless made a good recovery.
Dr. Gee	Aug. 22, 1874	M.	2½ "	1	...	Operation four hours after admission to the hospital.
Dr. Dickinson . .	.	Oct. 11, 1874	F.	6 "	1	...	Other cases had occurred in the same house; she had been ill eleven days; operated on a few hours after admission.
Dr. Dickinson . .	.	Nov. 2, 1874	F.	8 "	1	...	Had been ill one week.
Dr. Dickinson . .	.	Nov. 19, 1874	M.	2½ "	...	1	Antimonial treatment, then sulphate of zinc and ipecacuanha; child was comatose when operated on; never revived again; was made to breathe oxygen; died ten hours after operation.
Dr. Gee	Jan. 23, 1875	F.	2 "	...	1	The child died seventeen days after an operation under symptoms of general sepsis.
Dr. Dickinson . .	.	Jan. 30, 1875	F.	6½ "	1	...	Was convalescent from typhoid fever.
Mr. Thomas Smith .	.	March 4, 1875	F.	8½ "	...	1	Scald of glottis; diphtheria supervened on the sixth day.

Where operated on. Physician.	Date.	Sex.	Age.	Re- coveries.	Fatal.	Remarks.
<i>Hospital for Sick Children</i> (continued)—						
Dr. Dickinson . . .	April 1, 1875	M.	4 years.	1	...	Had been ill four days.
Dr. Chendle . . .	May 10, 1875	F.	3½ "	...	1	Vin. ipecac.; operation one hour after admission; died of erysipelas of neck and blood poisoning on third day.
Dr. West . . .	April 8, 1873	M.	5 "	1	...	Operation by Mr. Howard Marsh; after treatment by author.
Dr. Dickinson . . .	May 13, 1875	M.	3½ "	1	...	Operation by Mr. Howard Marsh; after treatment by author; boy still wears his tube.
<i>Private Operations.</i>						
Meredith Townsend, Esq.	April 13, 1875	M.	2½ "	1	...	Among other complications, had scarlet fever and nephritis; severe diphtheria.
Mr. Brend . . .	April 10, 1877	F.	11 "	...	1	Died of intense blood poisoning.
<i>East London Hospital for Children.</i>						
Author . . .	Sept. 17, 1878	F.	2½ "	...	1	Severe case; at the autopsy tubular casts were found in the small bronchi of both lungs.
Dr. Horatio Donkin . .	Sept. 25, 1878	F.	4 "	1	...	Operation by Mr. Hayward, House Surgeon; the case was complicated by some inflammatory oedema of neck.
Dr. Eustace Smith . .	Sept. 25, 1878	F.	2½ "	1	...	Operation by Mr. Hayward, House Surgeon; a cast of the trachea was got up at the operation.
Dr. Donkio . . .	Sept. 30, 1878	F.	9 months	...	1	Operation by Mr. Hayward, House Surgeon; child died suddenly; exact cause not known.

ON THREE CASES
OF
DISTAL LIGATURE OF THE CAROTID
AND SUBCLAVIAN ARTERIES
FOR
ANEURISMS INVOLVING THE INNOMINATE
ARTERY.

BY
RICHARD BARWELL, F.R.C.S.,
SURGEON TO, AND LECTURER ON SURGERY AT, THE CHARING CROSS HOSPITAL.

(Received November 7th—Read December 10th, 1878.)

On the 13th November, 1877, I read before this Society a paper on a case of large aneurism of the innominate, subclavian, and carotid arteries, for which I had performed distal deligation of the two latter vessels.¹ The man afterwards died of broncho-pneumonia, and in a subsequent communication, the parts were exhibited, showing complete obliteration of the vessels and almost complete obliteration of the sac. It will, perhaps, be remembered by those interested in such cases, that the part of the cavity still unoccupied by active clot had no channel through it, and therefore that its entire obliteration was a mere question of probably a very short

¹ 'Medico-Chirurgical Trans.,' vol. lxi, p. 13.

time, which, however, the man, with almost inconceivable folly, did not give himself. For the better elucidation of that matter, I have again placed on the table that preparation, and also because it may serve for the illustration of some points in the remarks that I have to make on three other cases of double distal ligature.

One of these cases, a man, æt. 48, sent to me by my friend, Dr. Cole, of Bath, underwent the operation on the 6th December, 1877. He died in about thirty hours, the thoracic portion of the aneurism being so large, and the power of respiration so restricted that he never recovered the anæsthetic (ether), but remained cyanosed and epileptoid to the end. The preparation from that man is on the table, but the case does not appear to contain material of such interest as to warrant me in taking up the time of the Society with its details. The operation on the subclavian was most difficult, for the vessel lay behind a clavicle so raised at both acromial and sternal ends, that I could do little more than reach it with the finger, and to get an aneurism needle round it was quite impossible. I managed, however, to pass the ligature by feeling behind the vessel for the eye of the needle, which could not be got beyond its lower edge, and then picking the catgut out with my fingernail. The man, however, lost hardly any blood.

On the same day I tied the carotid artery and the third part of the subclavian of Laura G—, and I beg here to thank my late colleague, Mr. Canton, for ceding to me that case, whose history is subjoined.

Laura G—, æt. 37, was admitted into Charing Cross Hospital on the 20th November, 1877, with an aneurism at the upper part of the right side of the chest.

Past history.—She is the only child of a mother who died in her twenty-fourth year, of phthisis. The father died, aged thirty-one, of an unknown cause. She, after having been a housemaid for three years, married at the age of twenty. Has had six children; two were stillborn, three died in early infancy (two of convulsions, one of bronchitis), the surviving girl is delicate, and has been under my care

with necrosis of the tibia. Laura G—'s husband died five years ago. She then took a coffee shop, where she found the task of putting up and taking down heavy shutters and other work try her so severely that she gave up the occupation in nine months. Two years ago, therefore two years after ceasing this sort of exertion, she first found her breathing easily disturbed, and about September, 1876, she first had indefinite, but sharp, shooting pains about the upper part of the chest. About eight months ago she first noticed a swelling "above the right breast." The indefinite chest pains then became localised in the right side and upper part of the chest, shooting also down the right arm, which was often numb; her breathing became embarrassed, sometimes even painful; she had a harsh, irritating cough (especially when she lay down) without expectoration, either of blood or mucus. During the last two months the swelling has markedly increased. She feels very faint when standing; she cannot lie without being much propped up by pillows, and sleeps only in snatches, being constantly awake by cough and a sense of suffocation.

State on admission.—Patient is a spare woman of cheerful disposition, but of highly nervous temperament, therefore, probably, some deductions have to be made in the account of her past sensations, especially as, though looking worn, she seems, save for the aneurism, in fair health.

A projecting tumour, with broad base and rounded apex, occupies the part of the chest lying between the lower edge of the second right costal cartilage and the sterno-clavicular joint; it protrudes above the episternal notch, and presses forward the right sterno-mastoid muscle. Its inner or left margin covers the right part of the sternum to the extent of one third of its breadth. It measures transversely one inch and three quarters; its lower edge lies two inches and a half below the sterno-clavicular joint, but just below this articulation a small portion of the chest-wall seems free of tumefaction. This tumour pulsates visibly; to the touch the pulsation is markedly expansile, and for a certain distance round the apex the impulse seems very near the surface.

Pulsation can also be felt through the chest-walls for a considerable distance round the tumour, viz. on the left clavicle for about an inch, on the right for an inch and a half from its inner end. If from these points two curved lines (convexity outwards) be drawn to the upper part of the third right costo-sternal articulation a very fair map of the pulsation-limit on the chest will have been obtained (see diagram,

FIG. 1.

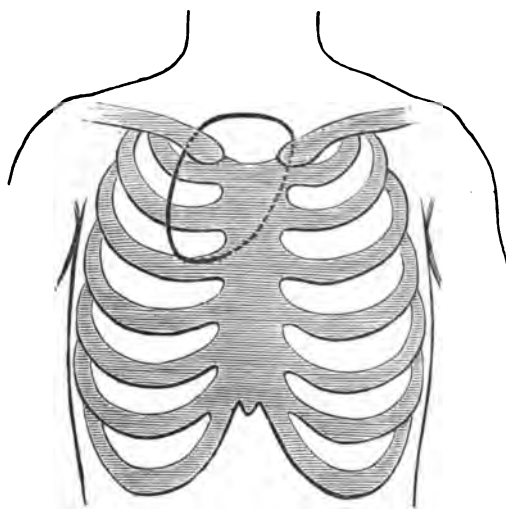


Fig. 1), but to this must be added the cervical pulsation, rising one inch and a quarter above the edge of the sternum, reaching under the inner edge of the left, and to the outer side of the right sternal portion of the sterno-mastoid muscle. Dulness to percussion mingled, on the left side of this space, with the cardiac dulness, but on the right was less extensive than the pulsation, as though the lung covered that portion of the aneurism. Over the whole area of this space the heart-sounds are loud, with a metallic ring, but the second sound is more especially remarkable as being even louder than the first, and having a peculiar, dull, yet metallic note.

Sphygmographic tracings of the left and right radial pulse were carefully taken by Mr. Wickers (figs. 2 and 3).

FIG. 2.



FIG. 3.



The patient was treated by diet and rest, but the tumour increased slightly, and the pulsation at its apex seemed approaching still nearer to the surface; therefore, on

December 6th, 1877, I tied antiseptically, and with cat-gut, the right common carotid artery close to its bifurcation, and the third part of the subclavian artery.

7th.—Patient passed a good night, but is somewhat sick from the ether, and complains of severe headache *not* limited to the right side. Her diet, which, on account of sickness, she takes in small quantities at a time, is No. 2.¹

8th.—Headache disappearing. The pulsation in the tumour itself appears more distant.

10th.—The wounds are almost healed. There is some diminution in the area and force of pulsation. She complains of a sense of numbness in the right arm.

14th.—The carotid wound has healed. The upper part of the pulsatile tumour appears solid, as also does that above the sternum. The size of that on the chest has diminished, it is now an inch and an eighth in transverse, and two inches in perpendicular, measurement. Her cough has almost ceased.

19th.—Clavicular wound has healed. No radial pulse.

22nd.—There is barely any pulsation in the episternal notch. She was put on a dry diet, namely, breakfast and supper, 1 egg, 4 oz. bread, 3 oz. milk, $\frac{1}{2}$ oz. butter; dinner,

¹ Breakfast and supper—milk, 1 pint; bread, 4 oz.; butter, $\frac{1}{2}$ oz. Dinner—broth, 1 pint; bread, 4 oz.

ptom save the comatose condition. The tumour is in the same state as on the 5th.

11th.—The patient has had no return of fainting, indeed, has recovered her usual condition.

18th.—During the last week the tumour has decreased in size, and the pulsation is less marked.

23rd.—The tumour has been steadily decreasing, and is now on a level with the chest-wall; the pulsation is less distinct. Slight radial pulse.

May 20th.—Nothing worthy of report save almost entire disappearance of pulsation has occurred. Some domestic trouble has greatly excited her, and again the tumour projects slightly, but the pulsation has not increased in a like ratio as it previously did, in fact, the sensation gives rather the idea of a solid tumour with communicated pulsation. On each occasion of increase ice-bags, in alternate six hours, were applied, and on each occasion as the excited condition of nervous system disappeared the tumour and increased pulsation also vanished. On June 13th was another but very slight exacerbation due to the advent of menses.

July 22nd.—Patient allowed to get up and move about a little, this has not increased the pulsation, and there is now no tumour.

August 15th.—Patient out for about three hours yesterday, and underwent an examination at one of the offices of convalescent homes. She came back very tired, but without increase of pulsation or appearance of tumefaction; nor at this date, the following day, can I detect any difference.

26th.—Patient left the hospital.

There is a part of the chest-wall roughly corresponding to the dimensions given in the first description of the case, which although it looks normal, or nearly normal, may be felt to be, beneath the skin, more protuberant than the same part on the other side. Here a distant communicated pulsation, not expansile, can be felt. A little below the sternoclavicular joint a small part about an inch in perpendicular, and two thirds of an inch in transverse direction, is dull to percussion. The rest is fairly clear.

On this case I would remark (further observations are to be found in the sequel) that the radial pulse was entirely absent for nearly four months—then occasionally a flicker was perceptible; afterwards a more uninterrupted pulse set in; and by the time she left the hospital, a weak, small, but constant right pulse was established.

The patient was in attendance to be examined by the Fellows of the Society. In the middle of February, 1879, she was married.

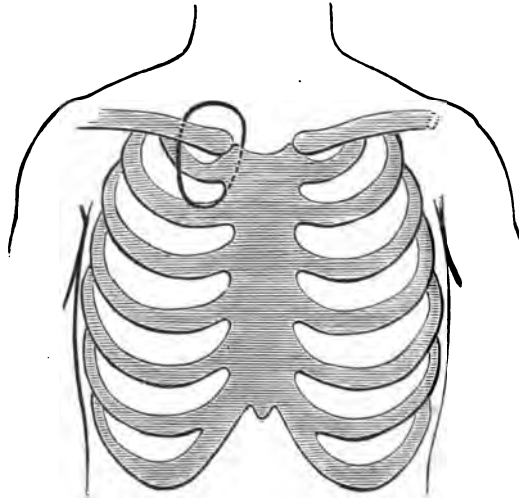
CASE 4.—Catherine H—, æt. 27, was placed under my care by my friend Dr. Julius Pollock on 10th January, 1878.

History.—She comes of a healthy family, but she, though not suffering from any disease, has never been strong, more especially since a severe attack of acute rheumatism fourteen years ago, which kept her in bed more than four, and in the house nearly six months. Since then, until disabled by her present malady, she has been a nurse, her hardest work being carrying children. A little before Christmas, 1876, she suffered from palpitation, breathlessness, and sickness, with pain at the upper part of the right chest; occasionally these troubles would be alleviated, but only for a short time. About March of last year a medical man noticed pulsation above the clavicle; she was taken into a cottage hospital and kept at perfect rest and on strict diet for two months; she was better and returned to her employment. In three weeks the pulsation returned worse than before, accompanied by breathlessness and cough, and she has been able to do nothing since.

Present state.—Patient is a slightly-built, spare, young-looking woman, whose general functions are well performed, except that she menstruates too frequently and too abundantly. She gets out of breath on very slight exertion, and has a constant teasing cough, worse when she lies down. On looking at her neck one is at once struck by a peculiar wide and distinct separation of the sternal and clavicular portions of the sterno-mastoid muscles, leaving above the clavicles two rather wide triangular intervals, in which

probably no muscular fibres exist. In the right interval a pulsating tumour, looking about the size of a cobnut, is visible. To the touch this tumour seems larger; its pulsation can be felt inside the sternal portion of the muscle, as also under the clavicular part, for a considerable distance. By turning the head so as to relax this muscle one can grasp the tumour between the finger and thumb, when its pulsation is felt to be remarkably expansile and its walls appear very thin. The pulsation limit just above the clavicle is an inch and three quarters broad and nearly an inch high; pulsation may also be felt beneath the clavicle, and after any slight exertion in the first intercostal space down to the upper border of the second rib. Percussion notes are dull on the inner two inches of the clavicle, and in a semi-circle beneath this part extending down to the second rib (Fig. 5). On all

FIG. 5.



this part the second sound of the heart is markedly loud, with a dull but somewhat ringing thud. Sphygmographic tracings of the two radial pulses were taken by Mr. Wickers (figs. 6 and 7).

FIG. 6.



FIG. 7.



January 17th.—I tied the common carotid a little below the bifurcation and the third part of the subclavian.

18th.—The patient complains of intense headache, chiefly referred to the vertex. The temperature of the right side of neck, of the right shoulder and arm, is normal; there is slight numbness of the fingers; the right pupil is normal.

21st.—The wounds, dressed to-day, are nearly healed, the tumour of the neck pulsates as before; the patient is menstruating before her proper time.

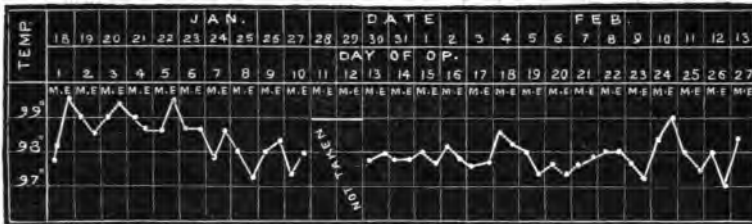
23rd.—The right radial pulse seems faintly perceptible. There is no brachial pulsation. The wounds are healed and the antiseptic dressings discontinued. A dry diet identical with that of L. G— was ordered.

29th.—The radial pulse is sometimes perceptible, at other times not so. The cervical tumour is less perceptible; it still pulsates, but appears more solid.

February 8th.—The tumour, which during the last six days has decreased both in size and force of pulsation, beats to day more violently. The patient is again menstruating and I may as well remark here that throughout her stay in the hospital, increase of pulsation always accompanied this function, which was both too frequent and too profuse. The cough has almost entirely disappeared.

16th.—The radial pulse is only occasionally perceptible; there is a good deal of pulsation at the proximal side of the carotid ligature.

FIG. 8.



20th.—Not very well, having some biliary derangement with sickness and headache. There is no cough nor other thoracic symptom; radial pulse faintly perceptible.

March 19th.—The patient has been going on very satisfactorily. The supraclavicular tumour considerably diminished in size, appears solid; it rises, however, with each pulsation, and its wedge-shaped bulk being forced up between the compressing fingers, gives a sensation as of expansile pulsation. I permitted her to go out to make arrangements about going into the country. She was absent about an hour. On her return she was much exhausted, and the pulsation below the seat of ligature was increased, and she complained of dragging pains from that place towards the heart. After resting in bed for an hour or two, all these sensations disappeared, and she regained her usual condition.¹

20th.—Pulsation of the tumour is still very marked; the right radial pulse is quite perceptible, but small. She is to keep her bed.

23rd.—The additional amount of pulsation has disappeared and she is now in the state reported in the early part of the month.

April 1st.—I examined this patient with very great care. All the pulsation about the chest wall has disappeared; percussion dulness reaches barely an inch outward from the sternal end of the clavicle and about half an inch below that bone. The heart sounds are too loud over a rather larger

¹ This description of the effects produced by her walk are *verbatim* from the registrar's notes.

space; the second sound is not especially well-marked. The tumour above the clavicle pulsates visibly and to touch; it is, when the sterno-mastoid muscle is relaxed, easily grasped between the finger and thumb and its pulsation feels, on a first impression, expansile; careful palpation of the part has however convinced me that the tumour does not expand on pulsation; it seems firm and solid and I feel the mass rise up from the chest at each beat of the heart. It is in fact a cone which being forced upward by each pulse between the finger and thumb acts like a wedge and gives a false sense of expansion. Although the tumour is greatly solidified, I doubt its being altogether solid.

July 22nd. The patient was virtually discharged at the end of May, but was kept in the hospital until a convalescent home could receive her. The tumour in the neck persisted and still pulsated in the manner above described. The heart sounds were more audible than they should be in the right upper chest. The ratio between first and second sounds was normal. She has no cough or other chest symptom, but she occasionally gets into a nervous or hysterical condition when she cries and says she has the same pain as before the operation; when not in this condition, she denies having any pain in the chest at all.

November 5th—C. H—, presented herself at the hospital and was carefully examined by Dr. Pollock and myself. She has no cough; no difficulty of breathing, but can run up and down stairs, or up hill with ease. The right upper chest and the clavicle are resonant on percussion; the heart sounds a little too plainly audible, are normal in their ratio and have no metallic ring nor thud which were prominent previous to operation. The radial pulse (right) is very small, there is no carotid pulse above the seat of ligature.

On looking at the root of the neck a rather wide pulsation is visible; the greater part of which is undoubtedly venous, but on feeling behind the sterno-mastoid a pulsatile tumour is perceptible, upon which I have no further remark to make than that in the report of 1st April, except that this tumour has not increased, but rather decreased in the interval. I

hope to be able to show her when this paper is read, and the fellows of the Society will be able to judge for themselves of the condition.

Remarks.—A very few words will suffice to say all that need be added to these histories. It appears that when women in the middle period of life are the subjects of aneurism, whether or not they undergo operation, many disturbing causes prevent such a fair and full estimate of progress or the reverse, as we can form if the subject be male. I had occasion to observe these peculiarities in the case of a lady with femoral aneurism some four years ago. I am not, however, aware that this so strongly marked excitement of the vaso-motor system during the menstrual period has been previously noted. The histories indeed of these cases show many fluctuations and vicissitudes, all of which, except when L. G. was frightened (and even then she was unwell), were connected with that function, and all of which were, I believe more apparent than real. That is to say, the increase of pulsation at certain times was due to increased action and motion of the whole vascular system, rather than to any increased size of the aneurism.

A few words must be said about organisable ligatures, for I think the advantages of being able to leave a ligature after metamorphosis, upon an artery have been hardly appreciated, nor has this ability had its due practical influence on our mode of deligation. Little or no advantage has been taken of the fact, that we can now occlude the tube without destroying the continuity of a blood vessel. Yet upon this characteristic of the organisable ligature, I believe its value and safety to depend. Ever since the final abandonment of the flat tape ligature, more especially since the classical work of Dr. Jones, "On the Suppression of Hæmorrhage," 1805, it has been the universal doctrine of surgery, that ligatures must be tied sufficiently tight, to divide both the inner and middle coats of the vessel. This is doubtless necessary when such ligatures as must come away, that is, such as must ulcerate through the vessel, are

used—partly because the retracted and corrugated inner coats adhering to the coagulum form one of the barriers to hæmorrhage, and partly because it is necessary to shorten the ulcerative process. But if an organisable ligature be used in this manner there will be, when the cord is dissolved or metamorphosed, nothing between the blood stream and the outer parts except the somewhat loosely constructed outer coat wrapped round with a narrow line of recently formed fibre tissue. Therefore, unless the sealing with coagulum have been rather more rapid and more firm than is usual with large vessels tied in continuity, there will be some risk of secondary hæmorrhage. I cannot but think that we must attribute to this cause such instances of that disaster as have been from time to time reported to sister-societies and in the medical journals. But if we so use the ligature that only the inner coat, or, better still, that no part of the artery be divided, the persistent ligature and the coagulum will keep the artery occluded, while the tough elastic middle coat, supported by the outer, will effectually prevent hæmorrhage.¹ In working out this idea I found by gradually increasing experiment that catgut ligatures were perfectly safe when tied comparatively loosely on the largest vessels divided in amputations. I also found that catgut (size E violin string) will cut through both inner coats of the carotid, subclavian and femoral arteries if tied with a pressure of 3 lbs., but either no coat or only the inner serous tunic if tied with a force equal to 2 lbs.² In all cases of deligation of vessels I have, judging only from the action of the hands, endeavoured to use less than this minimum force; applying the ligature with only sufficient power to check the blood-current in the parts beyond, thus avoiding division of the middle coat. Partly to this mode of using the ligature, partly to the use of antiseptic precautions, must be attributed my somewhat remarkable success in these operations.

¹ I must however, express a strong opinion that catgut is neither a reliable nor a well-chosen material.

² These experiments were made on a large number of fresh subjects, with a dynamometer in each hand.

Let me recapitulate the statistics given in my last paper. Six cases of simultaneous deligation of carotid and subclavian for innominate aneurism were quoted. The case I then recorded was the seventh, and was the only successful one. Since that paper was read Dr. Kelburne King, of Hull, has performed the same operation; his case is most interesting and instructive, but the patient (drunken and insubordinate) died from suppuration of the sac and hæmorrhage on the eightieth day.

The three cases which I this evening add to these statistics make up the whole number of such operations for innominate aneurism to eleven; of which four belong to me. Of the eleven cases three are successful; all these are mine.

¹ 'Lancet,' 8th June, 1878.

ON DISEASE OF THE MASTOID BONE.

BY

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THE objects of the following communication are, to add another case to those already recorded, in which a purely local irritation excited malignant disease in a patient with no discoverable predisposition towards it, and in which epithelioma in its progress eroded and destroyed large portions of bone; to call attention to the instances in which the probable fatal effects of inflammation may be averted by perforating the mastoid cells; and to illustrate how the whole of the temporal bone may, in a child, be destroyed by ulceration and the patient survive.

In March of this year, Agnes S—, a married woman, æt. 32, whilst picking her left ear with a hair-pin, ruptured the tympanic membrane, and soon after the accident came under my notice as an out-patient of St. George's Hospital. With the exception of this lesion she was, in all respects, in good health. The rupture did not heal, and in a few days from the fistulous opening thus established, there was a constant discharge. A month later, after an attack of

which was followed by facial paralysis of that side, I again examined the ear and found a polypoid mass filling up the cavity of the tympanum, the membrane having by this time quite gone. She now came into the hospital; I removed the polypoid growth, and the pains in the ear, which had previously been considerable, passed off. Her stay in hospital, on this occasion, was three weeks.

On July 31st, when she again applied for relief and was admitted, she stated that she had suffered from no further pain until within five weeks, when acute pain in the ear came on, and soon afterwards the parts over the mastoid process became swollen and tender. Two weeks ago, she said, the skin over the swelling broke down and a little bloody matter was discharged. The ragged wound at that time observable was the result, and from this wound had been coming ever since a quantity of watery, very foul-smelling discharge. The skin over the mass was bluish, the tissues were infiltrated, and the edges of the wound were everted and hard. In short, the disease was to all appearance malignant. There were no enlarged glands, neither was there any history, to be obtained, of cancer in her family. No loose bone could be detected, although a large surface of bone was exposed. From this time she rapidly wasted; the wound increased in size until it formed a large cavity discharging most offensive matter, and she died on November 12th, from exhaustion without any head symptoms or hæmorrhage. The following is the description of the post-mortem appearance given by Mr. Ewart, the curator of the museum. Beyond what is mentioned there was no disease throughout the body.

“The ulcer, which was nearly circular, had a diameter of three inches. It was deeply excavated in the centre in the shape of a funnel; the diameter of this central hole was two inches; it extended forward and inwards to a depth of two inches in the direction of the long axis of the petrous bone, and terminated close to the lining membrane of the mouth at a point just internal to the inferior maxillary articulation. The surface of the cavity was foul, dirty green in colour, it was

partly formed by the white necrosed remains of the petrous portion and the sloughing cartilage of the jaw. At the periphery of the ulcer the colour was that of proud flesh. The surface was everywhere irregular and knobby; this tuberculation was especially marked at the lower part where there was a considerable amount of fibrous and œdematous thickening. No distinct tumour could be made out. The bone was extensively destroyed and absorbed. The mastoid process, the external meatus, and tympanum, as also parts of the petrous portions of the temporal bone had entirely disappeared. Viewed from the intracranial aspect the petrous surfaces were normal, but the dura mater having been stripped, it was found that the jugular foramen had become enlarged by ulceration. The inferior maxillary articulation was disorganised, and the articular surfaces exposed. The bone surrounding the gap showed no new growth infiltration, but the granulations, whilst spreading as a thin layer over the squamous portion of the temporal bone had noticeably eroded the subjacent bone.

Under the microscope, the diseased tissue consisted mainly of vascular meshes containing numerous small cellular islands, the smallest of these groups consisting of from four to six cells, presented ill-defined characters and intermediate forms between epithelial cell and fibre. The larger groups were plainly epithelial; no intercellular substance intervened between the cells; the cells had a large oval nucleus containing nucleoli or granules, and otherwise resembled epithelial cells. A few birds' nests were found.

The case is worth attention, as forming one of a series in which the mastoid bone has become the seat of malignant disease, and in which in every instance there has been a local irritation preceding the malignant growth. Thus, with one exception in all the cases I find recorded (there are six in all) the tympanic cavity had been in a state of suppuration for a long or short period before there was any evidence of malignant growth. In two of the cases reported by Sir William Wilde, the one a woman aged 50, and the other a man aged 55, there had been a discharge from the ear

for many years, and for several years in the case of a boy aged 7. The same condition was present in the examples related by Mr. Toynbee. In the exception referred to, although the local irritation did not take the form of a discharge from the ear, there was a distinct local cause assignable, as the subject, a boy, in being knocked down by a cab, received a violent blow on the side of the head. As he is said to have suffered from great pain in the ear, followed by facial palsy, it seems probable that even in this case the tympanum was the seat of inflammation for some time before the appearance of malignant disease. The case was reported by Mr. Cooper Forster, in the 'Pathological Transactions' for 1850. Like other morbid growths of the middle ear, malignant tumours appear to arise from the mucous membrane which lines the tympanum, and the rule seems to be that this mucous membrane is the seat of supuration for some time before malignant disease begins. Now this rule also applies to cases of polypus, and did apply in the case of a round-celled sarcoma, which is reported in my lectures on diseases of the ear. In regard to the origin of malignant disease without any apparent predisposition, six out of the seven cases form a small collection for consideration. Let them be placed with some thousands of cases in which from similar causes the tympanic cavity becomes inflamed, and the membrane is ruptured. In some the perforation will heal; in others the opening will remain through life, the tympanum discharging purulent matter more or less, but no growth arising; in others the local irritation of the discharging surface will induce polypus from the tympanum; in a few others will induce by discharge passing through the external canal, bony enlargements, the so-called exostoses. In the early days of the new growths how little (within our knowledge) divides the malignant from the simpler form. In the very case under notice, there was a period when from the cavity of the tympanum there was seen to be what to all appearance was a simple polypoid growth. After this was removed there was another period which may be described as one of quiet,

lasting for over six weeks, and during which no active disease seemed to be going on. Indeed, the malignant aspect was not shown until after an attack of inflammation of the mastoid cells, which before this attack had remained healthy. The unknown force which determines the character of the disease remains unknown, and it is but a poor explanation to say that the subject must have had a predisposition to cancer, seeing how easily cases of malignant disease might be brought forward in abundance, in which equally, as with this woman, no such predisposition is to be found. To show that without doubt cancer is frequently inherited, of course in no way disposes of the question.

The resemblance of this case to one where an ordinary ulcer of the leg takes on (to use a common expression) malignant growth in the form of epithelioma, which finally erodes and destroys portions of the tibia is very striking. In both a suppurating surface exists for some time before the simple granulations are replaced by epithelioma.

To pass on to the inflammatory disease of the mastoid bone—although the course of inflammation in general proceeds from the pharynx up the Eustachian tube into the tympanum, and so on to the mastoid cells, these cavities within the mastoid process occasionally become primarily inflamed, whilst throughout the whole course of the inflammation the tympanic cavity remains healthy. In each and every case, however, the principal points of importance to decide are, when and in what manner should an escape be artificially (*i. e.* surgically) provided for pus within the mastoid cells. These two questions are full of interest, and upon correct answers given to them the life of the patient often depends. I suppose it will be admitted that, if in all cases where there is pus within the mastoid cells no attempt were made to provide an egress for it, a certain number of patients would die from further complications before the process of ulceration had effected the desirable opening. Under what circumstances then should the mastoid cells be perforated? When should the case be allowed to proceed

till the bone has so far become softened as to permit of the opening of the so-called abscess by the knife? An answer to the first inquiry may I think be found from considering the following case, which came under my notice in the summer of the present year :

On the 10th June a young man in vigorous health was, whilst on board ship, without any apparent cause, attacked with acute inflammation in the right tympanum. The pain was so intense, that he did not sleep (he said, in narrating his case) for five days ; at the end of which period, the tympanic membrane gave way and allowed the escape of a purulent discharge from the external ear. Although this was followed by great immediate relief, at intervals he continued to suffer from pain in the head, till the 25th, on which day I saw him for the first time ; on the previous evening he had a slight shivering fit. The discharge was then profuse, the constitutional disturbance was great, and he had lost over a stone in weight since the beginning of his illness. There was very slight tenderness on *deep* pressure over the mastoid process, but no redness or œdema. The treatment pursued consisted simply of leeches over the mastoid process, hot fomentations, and the ear was syringed frequently with warm water.

Matters continued much the same until the evening of the 9th of July, when he had a severe rigor. On the next day prolonged and deep pressure over the mastoid process caused considerable pain, and, for the first time, very slight œdema was noticeable. On this day (under ether) I made an incision over the mastoid bone through the periosteum (the bone was healthy), and bored through the bone into the cells. This took some time to accomplish as the bone was very thick. When the opening into the mastoid cells was completed, about half a drachm of thick pus escaped through the wound. For the first time since the 10th of June he slept well through the night. On the following day the complete communication between the tympanic cavity and the mastoid

cells, was shown by the facility with which he could (the mouth, nostrils, and external ear being firmly closed) blow through the external wound. In doing so a quantity of pus was expelled. Twice during each day he cleared the tympanum and mastoid cells in this way, and the discharge, now escaping through the wound, ceased to come from the ear. Suffice it to say that he recovered without a bad symptom.

No surgeon who saw this case would, I believe, doubt the propriety of the proceeding which was adopted. Mr. Warrington Haward, who was good enough to help me at the operation, was completely in agreement upon this point. It was happily not done too late, but should it not have been done before? I cannot but reflect that I have never, either in my own practice or in that of others, known the mastoid cells to be opened before it was needed; but that I have seen, and I suspect that many others have done so, cases in which this operation might have been performed with advantage. I believe that when the bone is unusually thick, we are apt to be deceived by the absence of superficial tenderness and early œdema. In children, of course, the case is widely different; superficial tenderness, redness, and œdema, are early symptoms, and often are present before there is any pus in the mastoid cells. In such instances I have sometimes cut down to the bone, broken into the cells (this with children is, of course, readily done), and been disappointed at not finding an immediate flow of pus. Disappointed without reason, inasmuch as on the next day pus has escaped in plenty through the wound, the tympanum has been relieved, the acute symptoms have subsided, and the death of the bone has been avoided.

The point, however, which I would strongly urge is that, when in all these cases an incision has been made on to the mastoid process and the bone is found to be healthy, the opening into the mastoid cells should be completed; and that when, in the case of adults, a distinct rigor has taken place, this proceeding should not be delayed; because if the symptoms of what is termed mastoid abscess are waited for, the

patient will at times die from cerebral abscess, meningitis, or pyæmia.

When, in the above case, the discharge secreted by the lining membrane of the tympanic cavity had an escape provided for it through the external wound, and so ceased to be poured out through the perforation of the tympanic membrane, the hearing power at once increased, showing that the tympanum shared the improved condition of the other parts. The chance of inflammation proceeding upwards to the cranial cavity was thus at once lessened. Briefly, then, in similar circumstance there are few reasons which will warrant delay, and many which should urge decision.

These remarks will of course equally apply, whether the process of inflammation owes its origin to any of the exanthemata, or to so-called catarrhal inflammation of the middle ear.

After all that has been said of the necessity for prompt measures in these cases, it seems out of place to contemplate even the possibility of what in actual practice is common enough, viz. the death of the whole or portion of the mastoid process, especially as these dead parts remove themselves by the almost unaided efforts of ulceration. The disasters to the auditory apparatus, to the portio dura, and to life, which occasionally form rude interruptions to this process, also occur frequently enough to require no comment except to say that, it is with a view of occasionally avoiding such interruptions, I venture to advocate surgical aid.

The subject of the third case, L. E. B—, was a child, æt. 1 year and 8 months, who had a discharge from the ear with perforation of the membrane since the age of 6 months, and pieces of bone had from time to time come from the external auditory canal. It is only necessary to say that, at the time she came under my observation, on 30th March, 1875, there was a profuse purulent discharge from the right ear, facial palsy of that side, total deafness, and a large fluctuating swelling over the mastoid process; that after the swelling had been freely laid open the dead bone under-

neath was not found to be loose, but that a month later, I removed with my finger and thumb a large piece of dead bone which included in its mass, what to all appearance, was all that remained of the temporal bone. After this the wound healed; the external ear sank deeply into the pit which was left; the child recovered its health, and is now well with the exception of the loss of nervous power which has been referred to.

OBSERVATIONS
ON THE
EFFECT OF DIET, REST, EXERCISE, ETC.,
IN CHRONIC NEPHRITIS.

BY
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THE patient on whom these observations were made has suffered from chronic phthisis for several years; and is known to have had albumen in the urine at least since October, 1876. On June 16th, 1877, he was seized with an attack of what might be called acute albuminuria commencing after a chill, with lumbar pains resembling those of lumbago, slight shivers, total loss of appetite, headache, and fever. There was no œdema; on one day there was repeated vomiting. On the day of the attack he had hæmoptysis to the amount of at least a tablespoonful, probably induced by hurrying to catch a train, the weather at the time being very warm. On the following day the signs of a localised pneumonia in the lower lobe of the right lung were discovered. The symptoms and physical signs of moderate acute pneumonia with albuminuria persisted for ten days, declining from about the seventh day. The patient remained in bed from June 16th till

June 27th. The urine was smoky for the first four days, and had a peculiar odour. It was very little, if at all, diminished in quantity, but loaded with albumen, and on one day of a specific gravity of 1.050. No blood-corpuscles or casts were discovered; and it may be mentioned that in a similar subsequent attack, the urine was very carefully examined by an experienced chemist for the colouring matter of the blood, but none was found, nor, *à fortiori*, any blood-corpuscles.

The patient has had several attacks of the same kind, though their true character was only recognised in 1877. The first (?) occurred in April, 1875; and there were others of short duration in August, 1875, July, 1876, and October, 1876. There was also an attack in May, 1877, in which the "smokiness" of the urine lasted exactly four days.

From these details it will be seen that the case has features of special interest apart from the experiments on which this paper is founded, but we do not propose to enter further into them at the present time.

First Series of Observations.—Ordinary diet and conditions.

In July, 1877, as an examination of the urine revealed a considerable amount of albumen, the patient was advised, (and we must here take the earliest opportunity of expressing our extreme obligation to Dr. Lauder Brunton for many most important suggestions most kindly and willingly given us), to have repeated analyses of his urine made, with a view to determine the variations, under ordinary diet and conditions generally, in the proportions of albumen and urea in it, and the amount of the latter passed in the twenty-four hours.

By *ordinary diet* is meant a moderate amount of meat, with vegetables, abundance of milk and farinaceous food, and no stimulants. By *ordinary conditions* is meant daily walking exercise, short of great fatigue, with rest in bed from about 10 p.m. to 7.30 a.m.

The results of these preliminary experiments for each day are embodied in Table I, but we give the means of the whole set of observations here.

1. *The mean total amount of urine passed was by day* = 740 cubic centimetres (8 days' mean), and *by night* 387 cubic centimetres (mean of seven days' observations); the urine passed with the daily motion not being collected, but probably amounting to at least 200 cubic centimetres more.

2. *The mean proportion of albumen was by day* one eighth, and *by night* $\frac{1}{13\frac{2}{3}}$, reckoned from the observations of eight and seven days respectively.

3. *The mean weight of urea* in grammes in 10 cubic centimetres of urine was '475 (four days' observations) *by day*; and '263 (three days' observations) *by night*.

The albumen was estimated by the usual clinical method, as it was impossible to use the accurate one of drying on a weighed filter over sulphuric acid *in vacuo* and weighing, on account of the time and labour involved. *The specific gravity* was unfortunately not taken at this time.

Second Series of Observations.—Milk and vegetable diet ; no meat ; ordinary conditions.

On July 24th, after the preceding observations of the urine had been carried on consecutively for ten days, during which the patient took only the ordinary moderate diet described above, the diet was further limited, and no meat allowed for three days. The only strictly nitrogenous food besides milk was toast for breakfast, and a little tart-paste at dinner. *Butter, bacon-fat, ham-fat, potatoes, carrots, cabbage, and rice pudding* were eaten freely. The results of this second series of observations are given in Table II, but they may be stated generally as follows, as compared with those of the preceding days.

1. *The mean proportion of albumen* for the three days, July 24th, 9 a.m., to July 27th, 9 a.m., was *by day* $\frac{1}{7\frac{2}{3}}$ (against one eighth on ordinary diet), and *by night* $\frac{1}{19\frac{2}{3}}$ (against $\frac{1}{13\frac{2}{3}}$ on ordinary diet).

2. *The mean weight of urea was by day* .193 grammes in 10 cubic centimetres of urine; against .2475 grs., on ordinary diet during the four preceding days (5 determinations *per diem*); and *by night* .24 grs.; against .263 grs. on ordinary diet).

3. *The mean total amount of urine passed was* 718 cubic centimetres *by day* (against 740 cc. on ordinary diet); and 413 cc. *by night* (against 387 cc. on ordinary diet).

On the whole, therefore, the results were not very decided either way, and they may be summed up thus:—The proportion of *albumen* was slightly higher by day than on the days when meat was allowed, and lower by night. The *urea* was decidedly diminished by day and slightly by night. The *volume* of urine was less by day and greater by night. It should be stated that a note, appended to the analyses of the second and third days, remarks that “the general impression to the eye on first boiling” was “that there was less albumen in the urine on these days.” The patient’s *temperature* was taken regularly at 8 a.m., 5 p.m., and 10 p.m., but presented no special abnormality. The *maximum* observed was 99° F. in the evening; and the *minimum* observed was 97° F. in the morning.

It should be further noted, with reference to later experiments with milk and non-nitrogenous diet, that moderate exercise was taken as usual during this second series of observations. The point of this statement will be clear when we come to speak of the influence of *rest*.

Third Series of Observations.—Ordinary diet and conditions.

From July 27th to September 15th, no further exact observations were made; but they were resumed and continued regularly from the latter date until October 8th, the albumen and urea being estimated and the specific gravity and volume of urine noted usually five times a-day. The results obtained will be found *in extenso* in Table III. For purposes of

comparison with the results of the experiments next to be related, a statement of *means* for the whole number of days of *ordinary diet* during this second period is also given in Table IV.

Fourth Series of Observations.—Experiments with absolute milk diet.

The albumen having ranged on September 15th and 16th between one fifth and one fourteenth, with a mean of one sixth on September 15th, and of one eighth on September 16th, with ordinary diet; the patient was put for two days, September 17th and 18th, on *absolute milk diet*, exercise being permitted. Four and a half pints of milk were taken on September 17th, when the albumen sank to one sixteenth and one thirtieth; and four pints on the second day, September 18th, the albumen being further reduced to one thirtieth, one fortieth, and a trace. No analyses of the urea were unfortunately made on the first day (17th), but on the first night (18th) the urea fell to .14 gramme in 10 cc.; on the second day (18th) the mean of five analyses gave .164 gr.; and the analysis for the second night (19th) .185 gramme. The specific gravity was also low, ranging from 1.006° to 1.010° on the two days, instead of 1.016° to 1.017°. On the other hand, as might have been expected, the volume of urine was large, being 940 cc. by day, and 385 cc. by night on the first, and 1190 cc. by day, and 610 cc. by night on the second day of observation. (The mean volume on ordinary diet had been 722 cc. by day and 484 cc. by night.)

The detailed statement of these results is furnished in Table V.

On the second day the patient complained of a sensation of unsatisfied hunger, and of sinking, and declared that he could not continue an absolute milk diet very long. We must, however, confess that the quantity of milk allowed was probably too small.

The general conclusion to be drawn from these experiments

is much in favour of an absolute milk diet. Considering that few patients in moderate health can tolerate a purely fluid diet for any length of time, the result of this series of experiments suggests that the diet in albuminuria should consist of milk, at any rate, as far as can be borne. In subsequent experiments which will be immediately related, ordinary diet *plus* water was tried against ordinary diet *plus* milk. The result, which was not in favour of the addition of milk to ordinary diet, but rather the opposite, would appear to indicate that, in order to produce a diminution of the amount of albumen in chronic Bright's disease, milk must be given not to *supplement*, but to *replace* to a certain extent, the articles of ordinary diet.

On September 19th, after discontinuing the milk and returning to ordinary diet, the albumen rose from a trace to one seventh and one eighth, in the hours from 8 a.m. to 10.30 p.m.; and to one sixteenth in the night of September 20th. From 8 a.m. on that day to 9.15 p.m. it ranged between one sixteenth and one sixth, the reason for the latter high figure being hereafter to be discussed. The specific gravity rose on September 19th and 20th, from 1.010° to 1.015° and 1.020°; and the urea from .164 to .256 (day of 19th), .29 (night of 20th) and .28 (day of 20th).

Fifth Series of Observations.—Experiments with excess of eggs in the diet.

On September 21st, the effect of an *excess of eggs* in the diet was tried and continued for two days. The following articles of food were alone permitted:—Eggs boiled, fried, and in the form of omelettes, fat bacon, baked apples, turnips, potatoes, brocoli, grapes, a little toast and bread, and milk in small quantities only as a drink.

The results of these observations, which are embodied in Table VI, may be stated generally as follows:—

On the first *day* the albumen between 8 a.m. and 9.15 p.m. ranged between one eighth and one twelfth, with a mean of

$\frac{1}{53}$; the urea represented a mean of .248 grs. per 10 cc.; and the mean specific gravity was 1.019°. The means of the same *night* till 8 a.m. were:—albumen, one fourteenth; urea, .26, and specific gravity 1.020°. During the second *day*, up to 10.15 p.m., the albumen ranged between one eighth and one eleventh, with a mean of $\frac{1}{53}$; the urea between .20 and .26, mean .225; and the specific gravity between 1.014° and 1.020°, mean 1.017°. In the *night*, till 7 a.m., the albumen was one thirteenth; the urea .28; and the specific gravity 1.020°.

Thus, the results of these two days' experiments are by no means in favour of a diet of eggs; and, as a fact, if compared with the mean results obtained by mixed ordinary diet, are, if anything, in favour of the latter, especially on the second day of observation. This will be more readily appreciated if the results observed under the two different kinds of diet are arranged side by side, as in Table VII.

Sixth Series of Observations.—Experiments with ordinary diet, with water in place of milk.

On September 24th, in order to compare together the effects of ordinary diet with water, of the same diet with milk, and of an absolute milk diet, the patient was ordered four pints of spring water, with meat, vegetables, bread, butter, and fruit *ad libitum*, during the twenty-four hours from 9 a.m. of the 24th, till 9 a.m. of the 25th. The results are given in Table VIII, from which it will be seen that during the *day* the albumen ranged between one fifth and one fifteenth, with a mean of one tenth; the specific gravity was 1.013°; and the urea .26 grs. per 10 cc.; and that during the *night*, the albumen was a trace; urea .16; and the specific gravity 1.010°. The comparison of these results with those obtained under ordinary diet with milk, and with an absolute milk diet is given in Table IX.

On the whole the result of the experiment with ordinary diet and water is, if anything, better than of that with ordinary

diet and milk, but both are decidedly inferior to the results obtained with absolute milk diet. The question wherein consists the intrinsic value of milk in a case like the present, must be left for others to answer. Is it that the proportions of the various elements are so accurately adjusted in this perfect food? or is it that assimilation is rendered easier by the dilution of its ingredients?

Seventh Series of Observations.—Experiments with non-nitrogenous diet.

From September 28th, 8 a.m., till the 30th, 8 a.m., an exclusively non-nitrogenous diet was tried, consisting of mashed potato, water-arrowroot, fried cabbage, boiled rice, fat bacon, butter, and water. It is unfortunate that the results of the first day's experiments are to some extent vitiated by the fact that part of the urine was lost, owing to two watery stools passed in the forenoon after a seidlitz powder taken to relieve constipation and headache. The results of this series of observations are embodied in Table X.

The means of the two days, compared with the means of the combined observations of ordinary diet, are contained in Table XI.

A comparison of these figures clearly indicates a reduction in the amount of albumen excreted on the days when no nitrogen was ingested. It appears to be a further interesting fact, that the effect of the withdrawal of nitrogenous food on the excretion of albumen *does not cease with the restoration of nitrogen to the diet, but persists for a certain period afterwards*. Thus when nitrogen had been restored on September 30th, the urine of the morning collected at 1 a.m. contained a trace only (recorded as one one-hundredth), that of 4.15 p.m. one sixteenth, and that of 8.45 p.m. one fifteenth. Next day, at 11 a.m., one thirtieth albumen was registered, at 2.10 p.m. one fourteenth, and at 4 p.m. one tenth. These observations are set forth in Table XII.

This behaviour seems to be explicable on the assumption that the albumen which passes out of the blood into the urine is, ordinarily, in part at least, the residue of a *luxus*-consumption of nitrogenous food. Hence, when nitrogen is withdrawn for a time, and then restored, the organism has to make good its losses during the period of abstinence, and the albumen does not reappear in the urine in its original quantity until this process is more or less completed.

Eighth Series of Observations.—The effect of rest.

Still more interesting and important than the above observations were the results obtained by placing the patient in the recumbent posture for twenty-four hours, in a state of nearly perfect rest. These are embodied in Table XIII; and in Table XIV will be found the mean amounts of albumen, &c., from noon to noon, of October 1st and 2nd—the day preceding that on which the patient rested; of October 2nd and 3rd—the day of rest; and of October 3rd and 4th—the day following the period of rest.

A complete account of the state of the urine during the three days following the day of absolute rest is given in Table XV.

The results of the experiment on rest stated in words are as follows:—1st. The albumen was lower (apparently) than on any other day on which observations were made. 2nd. The amount of urine passed was greater than on any other day observed, viz., 1090 cc. 3rd. The urea by night was less than normal.

The diminution in the excretion of albumen during rest in this experiment receives support from the observations made on the same patient as to the reduction of the same excretion during the night. A comparison of the relative excretion of albumen during the two periods of day and night invariably show a marked diminution during the latter period; in fact, the mean excretion was occasionally as high as one seventh during the day, and only a trace at night.

one-hundredth) in the subsequent night; and, though the reduction might in the latter case be partly due to the albumen circulating since the last meal at 8 or 9 o'clock having already been in part excreted with the last urine passed before going to bed, so that the remainder would be excessively diluted by the large quantity of urine voided during the night, this argument would be scarcely applicable to an experiment like the above, during which the patient was taking his regular meals, and digestion was going on, and yet scarcely any albumen passed out of the blood. The observation that lying *in bed* has a beneficial effect on cases of chronic desquamative nephritis was made long ago by Dr. Bright ("Cases and Observations illustrative of Renal Disease," 'Guy's Hospital Reports,' April, 1840, p. 160), and has lately been confirmed by Bartels ("Krankheiten des Harnapparates," Ziemssen's 'Handbuch,' Band 9, zweite Auflage, 1877, s. 363); though the latter does not regard the rest as the cause of the benefit derived, but ascribes it to the uniform warmth of the bed, which by dilating the vessels of the skin relieves the renal circulation. The patient in the above experiment did not, however, lie in bed during the first part of the twenty-four hours from noon to bedtime, 10 p.m., October 2nd, but lay on a sofa in his clothes without being kept particularly warm. Moreover, repeated observations in this case have shown that exertion increases the amount of albumen; so much so that it can be predicted with almost absolute certainty that a long walk or much bodily exertion will nearly double the amount of albumen excreted. The effect not only of exercise, but also of food and cold upon the amount of albumen in Bright's disease, has attracted the attention of that excellent observer, Dr. George Johnson, though he only alludes to it in a general manner in the following passage, which was not known to us when our experiments were made. He says ('Lectures on Bright's Disease,' 1873, p. 131, Lecture 7):—

"A convincing proof and illustration of the effect of exercise, food, and cold upon the amount of albumen in the urine is afforded by the fact that in most cases of albuminuria, the

urine passed after rest in bed and before breakfast contains much less albumen than that secreted after exercise in the open air and after an ordinary meal." In Virchow's 'Archiv,' Band lxxii, Heft 2, Leube has lately described some examinations of the urine of a number of soldiers, where in a few cases it was found that albumen appeared *after severe marching* and disappeared again in a few hours.

Dr. Lauder Brunton too, has, in a valuable paper in the 'Practitioner,' on "Arsenic in Albuminuria," June, 1877, reported the case of a patient who sometimes only had albuminuria when he exerted himself much, or when he did mental or bodily work *in the morning*, while in the afternoon he could do similar work without bringing on the albuminuria.

Hence our case is by no means an isolated one as far as the effect of exercise upon the albuminuria is concerned, and we can well understand that if exertion will sometimes cause albumen to appear in apparently healthy men, *à fortiori* it will be more likely to do so in cases where the kidneys are manifestly diseased. A discussion of the *modus operandi* of exercise on the renal circulation would be out of place here, but the experiments of Stockvis should make us very careful before we explain it by increased arterial pressure. In any case, only a partial explanation of the phenomenon seems at present possible, and we have referred to the subject at such length here, chiefly with a view to call the attention of the profession to the importance of rest as a therapeutic measure in albuminuria, a measure which it appears to us has been partly underestimated and partly misunderstood.

Ninth Series of Observations.—Experiments with digitalis.

To see whether increased arterial tension affected the excretion of albumen, on October the 6th, *infusion of digitalis* was given to the patient. Much stress is not laid on the result, as, although the pulse was reduced from 92 to 72, the increase in the amount of albumen observed was not so great as to be certainly due to the effect of the *digitalis*.

result of these experiments will be found in Table XVI. We also give the figures for several days without digitalis, for comparison, in Table XVII.

Whether in this experiment the albumen was really increased by the digitalis or not, there can be very little doubt that the amount of urine secreted, 1420 cc., was greater than on the three previous days and the day after. As giddy feelings were complained of by the patient, and for other reasons, it was thought better not to press the digitalis very hard, or to continue it longer.

General Conclusions from the whole Number of Observations.

To sum up these experiments, we seem justified in arriving at the following conclusions in this case:—

1st. That the amount of albumen was reduced by absolute milk diet, and by absolute non-nitrogenous diet.

2nd. That the effect of the milk diet was not merely apparent, and due to the albumen being more than ordinarily diluted, for ordinary diet with an equal quantity of water did not produce the same result.

3rd. That eggs in excess did not appreciably reduce the amount of albumen.

4th. That the effect of a non-nitrogenous diet was not immediate, and that it persisted some time after nitrogen was reingested.

5th. That absolute rest remarkably reduced the amount of albumen; and,

6th. That increase of arterial tension by digitalis possibly increased its excretion. (See, however, *note in Appendix.*)

There are three points on which a few words still remain to be said. They are:—

1. The effect of abstinence from meat in the second series of observations, compared with later experiments on an exclusive diet of milk and vegetables and bread, carried out in November and December, 1877, and set forth in Table XVIII.

2. The effect of wine.
3. The effect of exercise.

1. *The effect of non-nitrogenous diet.*—It will be remembered that in the second series of observations meat was excluded from the diet, with the result that the proportion of albumen was somewhat higher ($\frac{1}{7.3}$) by day than the mean (one eighth) of ordinary diet; and decidedly lower by night ($\frac{1}{19.3}$ against $\frac{1}{13.3}$). In the later similar prolonged observations the amount of albumen has been much less, as the results of seven consecutive days, contained in Table XVIII, will prove. At the same time the patient took only the most gentle exercise, except in the instances to be mentioned under the head of exercise. Are the different results in July and December to be explained (a) by less exercise being taken and more rest in the recumbent posture; (b), by the beneficial effect of milk and vegetable diet not being apparent in the first case owing to too short a trial; or, (c), to general improvement in the state of the kidneys?

2. *The effect of wine.*—On July 21st, the albumen had ranged for two days at $\frac{1}{9.15}$. After two glasses of Moselle at dinner (6.30 p.m.) the amount suddenly rose to one fifth; the next morning at 6 a.m. it was one eighth. During the analyses of the urine of *ordinary diet* in the first series of observations, this is the only instance of so high a figure.

On September 20th, at 4.45 p.m., the albumen was one tenth. After half a glass of port wine it was one sixth at 9.15 p.m. At 7 a.m. next day it was one sixteenth.

On September 25th two glasses of champagne were drunk at 7 p.m. At 10.30 there was one eighth, and in the urine of the night to 8 a.m. one twelfth albumen.

Similarly, half a glass of Madeira at 7 p.m. was followed by one fourth albumen at 8.30 p.m., there having been only one tenth at 4 p.m., and one sixteenth in the subsequent night.

On the other hand, it is to be noticed that although no

wine had been taken, one fifth was noted on July 24th, on September 15th and 24th (2.15 p.m. water only drunk), and October 4th (after curry); while on September 29th, after one glass of Madeira at 6.30 p.m., the albumen was only one fifteenth instead of one sixteenth as at 2.30 p.m. and 6.30 p.m. (but non-nitrogenous diet); and on September 30th, after half a glass of Madeira at 1.30 a.m., one sixteenth (ordinary diet).

3. *The effect of exercise.*—So very little severe exercise was taken during the first and second series of experiments, that it is only occasionally that any decided effect from it can be traced.

On September 24th, however, after half an hour's walk and much standing all the morning, the albumen rose suddenly to one fifth, falling at 5.30 p.m. to one fifteenth, and at 9.45 p.m. rising again to one twelfth.

In some later observations in which the diet was limited to milk and vegetables for some weeks, the effect of exercise was much more decided. Thus, on November 21st, after a tiring walk of one hour and a half, with climbing a steep ascent the albumen rose to one seventh at 9 p.m., after it had ranged for many days between one twelfth and a trace.

On December 2nd, after going to morning-church and standing a good deal, one eighth was noted at 2.20 p.m., the average amount at the same hour on many days before and after being one fifteenth to a trace.

On December 9th, under similar circumstances, the albumen, which was one fourteenth at 10.40 a.m., rose after church to one ninth at 2 p.m., and fell at 5.30 p.m. to one twenty-eighth, and at 9 p.m. to one sixteenth.

Whether these variations are really due to the exercise taken we do not presume to say definitely. They certainly however agree with the other observations in this particular case, and with those of the authorities referred to earlier in our paper.

APPENDIX.

Note to paragraph 6, page 254.—On the Effect of Digitalis upon the Excretion of Albumen.

We should not at first expect the albumen to be increased by raising the arterial blood-pressure with digitalis. Venous congestion is (according to Stockvis) the cause of albuminuria depending on alterations in the circulation. (Brunton, l. c., p. 430.) But digitalis may cause more albumen to appear by first lowering the blood-pressure in the glomeruli owing to spasmodic contraction of the renal arterioles through some specific influence on the latter, which Brunton and Power have discovered to exist. ('Proceedings of the Royal Society,' 1874, No. 153.) This lowered blood-pressure is attended with a diminished secretion, or even arrest of the secretion, of urine; and there has been found to be a general inverse relation in albuminuria between the amount of urine secreted and the amount of albumen (Brunton, l. c., and our own observations), so that when, as in renal disease, albumen is being continually excreted, if the amount of urine is diminished by digitalis, *à fortiori*, the amount of albumen will possibly be increased.

In the following charts an attempt is made to represent graphically the absolute amount of albumen passed per hour, during the various series of observations. The values in these charts have been obtained by multiplying the total volume of urine passed during (say) the day by the proportion of albumen, and dividing the result by the number of hours. For example, on July 16th the absolute amount of albumen passed per hour was—650 cc. (urine) $\times \frac{1}{8}$ (albumen) $\div 14.5$ (hours), = 5.600 cc.

CHART I.—Showing the absolute amount (by volume in cubic centimetres) of albumen passed *per* hour, day and night, (1) under ordinary conditions of diet and exercise, during the eight days and eight nights *preceding* the first series of experiments—July 16—23 inclusive; and (2) under ordinary conditions of exercise, but on a milk and vegetable diet only, during the four days of the first series of experiments.

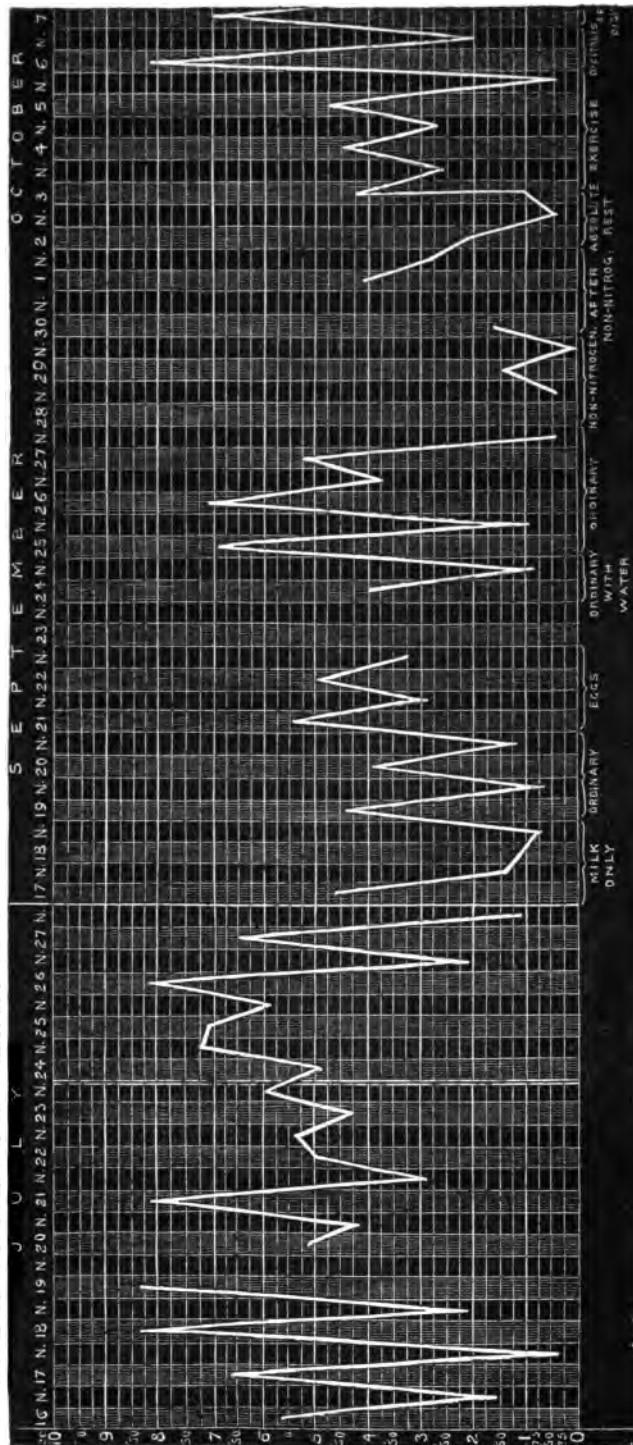


CHART II.—Showing the absolute amount (by volume in cubic centimetres) of albumen passed *per* hour, day and night, during the various subsequent experiments, arranged chronologically.

The varieties of diet and of conditions are indicated at the bottom of the chart.

TABLE I.—*Showing the results obtained on ordinary diet and under ordinary conditions. First time.*

Date.	Total urine in cc.		Urea in 10 cc.		Albumen, mean of.		Sp. gr.	
	Day.	Night.	Day.	Night.	Day.	Night.	Day.	Night.
1877 July 14.....	† nearly	† 1/2	Not deter- mined	Not deter- mined
" 15.....	†	1/2	"	"
" 16.....	650	200	†	1/2	"	"
" 17.....	860	320	†	Trace	"	"
" 18.....	960	400	†	1/2	"	"
" 19.....	840	No de- termi- nation	†	Trace	"	"
" 20.....	665	400	·31	...	†	1/2	"	"
" 21.....	875	480	·26	·28	1/2	1/2	"	"
" 22.....	650	400	·20	·30	†	†	"	"
" 23.....	430	510 (mixed urine) *	·22	·21 (mixed urine) *	†	† (mixed urine) *	"	"

TABLE II.—*Showing the results obtained on a nearly non-nitrogenous diet and milk and under ordinary conditions.*

Date.	Total urine in cc.		Urea in 10 cc.		Albumen, mean of.		Sp. gr.	
	Day.	Night.	Day.	Night.	Day.	Night.	Day.	Night.
1877 July 24	495	620*	·19	·27*	†	†	—	—
" 25	825	540*	·19	·24*	†	1/2	Not de- termined	Not de- termined
" 26	835	390	·20	·22	†	1/2	"	"
" 27	760	320	·24	·26	†	1/2	"	"

* Mixed urines of 10 p.m. and night.

† Last urine at night mixed with that of the night,

TABLE III.—*Showing the results obtained on ordinary diet and under ordinary conditions. Second time.*

Date.	Total urine.		Urea in 10 cc.		Albumen, mean of.		Sp. gr.	
	Night.	Day.	Night.	Day.	Night.	Day.	Night.	Day.
1877								
Sept. 15.....	—	—	—	—	—	‡	—	—
„ 16.....	—	—	—	—	†	‡	†	1015
„ 19.....	610	500	·18*	·16	Trace§	‡	1010	1015
„ 20.....	181	610	·29	·28	‡	‡	1020	1017
„ 25.....	765	900	·16	·21	Trace	‡	1010	1011
„ 26.....	420	820	·24	·24	‡	‡	1020	1017
„ 27.....	450	760	·30	·24	Trace	‡	1020	1016
„ 30.....	300	630	·20	·20	Nil	‡	1020	1014
Oct. 1.....	410	830	†	·20	†	‡	1020	1015
„ 3.....	†	†	†	†	†	†	†	†
„ 4.....	410	600	·28	·24	‡	‡	1020	1019
„ 5.....	450	920	·29	·27	‡	‡	1020	1018
„ 7.....	610†	790	—	—	‡	‡	1015	1017
„ 8.....	440	—	—	—	‡	†	†	†

* After absolute milk diet.

† No determination.

‡ After digitalis.

§ Traces reckoned as $\frac{1}{10}$.TABLE IV.—*Giving the means of the results obtained in TABLE III.*

Albumen.		Urea in 10 cc.		Specific gravity.		Total urine.	
Day.	Night.	Day.	Night.	Day.	Night.	Day.	Night.
$\frac{1}{6\frac{1}{2}}$	$\frac{1}{33\frac{1}{3}}$	·226 grm.	·242 grm.	1016	1017	722	484
12 days' observations	11 days' observations	9 days' observations	—	11 days' observations	10 days' observations	10 days' observations	10 days' observations

Note.—The mean excretion of albumen is lower in the second than in the first series of observations on ordinary diet; and the secretion of urine is somewhat higher by night. Does this point to improvement in the condition of the kidneys?

TABLE V.—*Showing the results obtained from a diet of milk only (Sept. 17th and 18th to 8 a.m. 19th).*

Date.	Quantity of milk taken.	Albumen.	Sp. gr.	Urea in 10 cc.	Quantity of urine in cc.	Remarks.
Sept. 17 (from 8 a.m.)	4½ pints	$\frac{1}{16}$, $\frac{1}{16}$	1008, day; 1010, night	No determination	Day = 940; night = 385	...
Sept. 18	4 pints	$\frac{1}{30}$, $\frac{1}{40}$, to a trace	1006, day; 1010 night	Mean = .164 grms.; Maximum = .185 grms.; minimum = .14 grms.	Day = 1190	Sensation of unsatisfied hunger and sinking
Sept. 19 (7 a.m.)	...	Trace	1010	.185 grms. mean of night	Night = 610	...

TABLE VI.—*Showing the results obtained on a diet consisting largely of eggs, fried, boiled, and in omelette; along with the following other articles, viz. fat bacon, baked apples, potatoes, brocoli, turnips, grapes, a little toast and bread, with milk as a drink.*

Date.		Mean of albumen.	Urea in 10 cc.	Sp. gr.	Total urine in cc.
Sept. 21	8 a.m.—9.15 p.m.	$\frac{1}{8}$ — $\frac{1}{12}$.248 grms.	1019	575*
„ 21, 22...	9.15 p.m.—8 a.m.	$\frac{1}{12}$.26 „	1020	430
„ 22	8 a.m.—10.15 p.m.	$\frac{1}{8}$ — $\frac{1}{11}$.20-.26 „	1014—1020	640
„ 22, 23...	10.15 p.m.—7 a.m.	$\frac{1}{12}$.28 „	1020	370

* Urine passed with stool before breakfast was not measured and is not included.

TABLE VII.—*Showing the mean results obtained under egg diet as compared with the mean results under ordinary diet.*

		Albumen.	Urea.	Sp. gr.
1. Egg diet.....	Sept. 21 (day)	$\frac{1}{2}$	·248	1019
Ordinary diet	Mean of many days	$\frac{1}{2}$	·226	1016
2. Egg diet.....	Sept. 21—22 (night)	$\frac{1}{2}$	·260	1020
Ordinary diet.....	Mean of many nights	$\frac{1}{2}$	·242	1017
3. Egg diet.....	Sept. 22 (day)	$\frac{1}{2}$	·225	1017
Ordinary diet.....	Mean of many days	$\frac{1}{2}$	·226	1016
4. Egg diet.....	Sept. 22—23 (night)	$\frac{1}{2}$	·280	1020
Ordinary diet.....	Mean of many nights	$\frac{1}{2}$	·242	1017

TABLE VIII.—*Showing the results obtained on ordinary diet with water instead of milk.*

Date.	Water drunk.	Albumen.	Sp. gr.	Urea.	Amount of urine in cc.
Sept. 24— Day to 9:45 p.m....	4 pints	$\frac{1}{2}$, $\frac{1}{2}$, $\frac{1}{2}$, $\frac{1}{2}$	1013	·26 grms.	570
Night	Trace	1010	·16 „	765

TABLE IX.—*Showing the comparative results obtained under ordinary diet with water, ordinary diet with milk, and absolute milk diet.*

	Albumen.		Urea.		Specific gravity.		Urine in cc.	
	Day.	Night.	Day.	Night.	Day.	Night.	Day.	Night.
Ordinary diet with water...	$\frac{1}{16}$	Trace	·26	·16	1013	1010	570	765
Ordinary diet with milk ...	$\frac{1}{8\frac{1}{2}}$	$\frac{1}{23\frac{1}{2}5}$	·226	·242	1016	1017	722	484
Absolute milk diet.....	$\frac{1}{16}$	Trace	·155	·185	1006	1010	1190	610

TABLE X.—*Showing the results obtained on a non-nitrogenous diet, consisting of mashed potato, water-arrowroot, fried cabbage, boiled rice, water, fat bacon, and butter.*

Date.	Albumen.	Sp. gr.	Urea.	Amount of urine in cc.	Remarks.
Sept. 28.—					
Day	$\frac{1}{26}, \frac{1}{16}$ mean = $\frac{1}{18}$	1011	·236	Not determined accurately	Two watery stools after seidlitz powder.
Night	Trace	1015	·24	450	
Sept. 29.—					
Day	$\frac{1}{16}, \frac{1}{15}$ Mean = $\frac{1}{15\frac{1}{2}}$	1015·5	·25	315	
Night	None	1020	·20	300	

TABLE XI.—*Showing the means obtained under a non-nitrogenous diet as compared with the means under ordinary diet with milk.*

Date.	Albumen.	Sp. gr.	Urea.	Amount of urine in cc.
Sept. 28.—Non-nitrogenous diet.				
Day	$\frac{1}{18}$	1011	·236 grms.	—
Night	$\frac{1}{100}$	1015	·24 „	450
Sept. 29.—Non-nitrogenous diet.				
Day	$\frac{1}{16.5}$	1015	·25 „	315
Night	No albumen	1020	·20 „	300
Average of many days, on ordinary diet with milk.				
Day	$\frac{1}{2.5}$	1016	·226 „	722
Night	$\frac{1}{23.53}$	1017	·242 „	484

TABLE XII.—*Showing the results obtained when the patient had returned to ordinary diet, on the days following the non-nitrogenous diet, and indicating the probable persistence of the effect of the latter.*

Date.	Albumen.	Sp. gr.	Urea.	Amount of urine in cc.	Remarks.
Sept. 30—					
Day	trace, $\frac{1}{16}, \frac{1}{16}$	1014	·20	630	...
Night ...	No determination	1020	No determination	410	...
Oct. 1—					
Day	$\frac{1}{30}, \frac{1}{10}, \frac{1}{4}$	1016	·21	...	Albumen $\frac{1}{4}$ was after wine
Night ...	$\frac{1}{18}$	1019	·29	450	...

TABLE XIII.—*Showing the results obtained under the conditions of absolute rest and ordinary diet from noon, Oct. 2, to noon, Oct. 3.*

Date.	Albumen.	Sp. gr.	Urea in grms.	Quantity of urine in cc.
Oct. 2—				
Day	$\frac{1}{18}$, $\frac{1}{40}$, trace	1013	·22	1090
Night	Bare trace	1011	·20	430
Oct. 3—				
Day to noon	Trace	1013	·21	410

TABLE XIV.—*Showing the mean amounts of albumen from noon to noon of the day preceding the day of absolute rest, of the day of rest, and of the day following the day of rest.*

Date.	Albumen.			Urea.			Specific gravity.			Total urine in cc.		
	Day.	Night	Day.	Day.	Night	Day.	Day.	Night.	Day.	Day.	Night.	Day.
Oct. 1—2	$\frac{1}{8\frac{1}{2}}$	$\frac{1}{18}$	$\frac{1}{40}$	·21	·29	·19	1016	1019	1012	630	450	160
„ 2—3	$\frac{1}{8\frac{1}{2}}$	$\frac{1}{100}$	Less than $\frac{1}{100}$	·21	·20	·21	1013	1011	1013	930	430	410
„ 3—4	$\frac{1}{10\frac{1}{2}}$	$\frac{1}{16}$	$\frac{1}{11}$	·20	·28	·20	1016	1020	1017	440	410	160

TABLE XV.—*Showing the results obtained on ordinary diet with exercise ; for comparison with TABLE XIII.*

Date.	Albumen.	Sp. gr.	Urea.	Amount of urine in cc.
Oct. 3—				
Day from noon	$\frac{1}{15}, \frac{1}{10}, \frac{1}{5}$	1016	·20	440
Night to 8 a.m.	$\frac{1}{15}$	1020	·28	410
Oct. 4—				
Day to 9.30 p.m. ...	$\frac{1}{15}, \frac{1}{11}, \frac{1}{5}, \frac{1}{5}$	1020	·24	600
Night to 8.30 a.m....	$\frac{1}{15}$	1020	·29	450
Oct. 5—				
Day to 10.30 p.m. ...	$\frac{1}{10}, \frac{1}{15}, \frac{1}{10}$	1017·5	·27	920
Oct. 6—				
Night to 8 a.m.	Trace	1016	·28	440

TABLE XVI.—*Showing the results obtained from the administration of digitalis, with exercise and ordinary diet.*

Date.	Hour.	Albumen.	Sp. gr.	Inf. digitalis.	Quantity of urine in cc.	Pulse.	Urea.
Oct. 6	11 a.m.	$\frac{1}{5}$	1014	2 drachms.	110	92	*
"	2.30 p.m.	$\frac{1}{5}$	1012	—	280	85	*
"	3.40 p.m.	—	—	1½ drachm	—	88	*
"	5.30 p.m.	$\frac{1}{5}$ scarcely	1018	—	180	76	*
"	10.30 p.m.	$\frac{1}{5}$	1015	1½ drachm	240	76	*
"	Night till 4 a.m.	$\frac{1}{10}$	1015	—	300	—	*
"	Night 4—8 a.m.	$\frac{1}{10}$	1015	—	310	—	*
Oct. 7	10.45 a.m.	$\frac{1}{5}$	1016	—	110	—	*
					Total 1420		

* signifies—No determination.

TABLE XVII.—*Showing the comparative results obtained without and with digitalis.*

Date.	Albumen.		Urine in cc.	
	Day.	Night.	Day.	Night.
Oct. 3 . .	$\frac{1}{12}$, $\frac{1}{10}$, $\frac{1}{8}$	$\frac{1}{8}$	440	410
„ 4 . .	$\frac{1}{10}$, $\frac{1}{11}$, $\frac{1}{8}$, $\frac{1}{8}$	$\frac{1}{8}$	600	450
„ 5 . .	$\frac{1}{10}$, $\frac{1}{12}$, $\frac{1}{10}$	$\frac{1}{10}$	920	440
WITH DIGITALIS.				
„ 6 . .	$\frac{1}{8}$, $\frac{1}{8}$, $\frac{1}{8}$, $\frac{1}{8}$	$\frac{1}{10}$	810	610
„ 7 . .	$\frac{1}{8}$, $\frac{1}{8}$, $\frac{1}{8}$, $\frac{1}{8}$	$\frac{1}{17}$	790	440

TABLE XVIII.—*Showing the results obtained as regards the proportion of albumen on milk diet with vegetables from December 1st to 8th.*

Date.	8 a.m.	Noon.	2.30 p.m.	5 to 6 p.m.	7.30 p.m.	9 to 10 p.m.
Dec. 1	*	Trace	$\frac{1}{16}$	$\frac{1}{16}$	*	$\frac{1}{16}$
„ 2	*	$\frac{1}{16}$	$\frac{1}{16}$ after church	$\frac{1}{16}$	*	$\frac{1}{16}$
„ 3	*	Cloud	Cloud	$\frac{1}{16}$	Cloud	$\frac{1}{16}$
„ 4	Faintest cloud	Faint cloud	„	Faint cloud	*	$\frac{1}{16}$
„ 5	„	Trace	*	Trace	$\frac{1}{16}$	Faint trace
„ 6	Faint cloud	$\frac{1}{16}$	Faint cloud	$\frac{1}{16}$	*	Trace
„ 7	*	$\frac{1}{16}$	$\frac{1}{16}$ (4 p.m.)	$\frac{1}{16}$	*	$\frac{1}{16}$
„ 8	Faint haze	Cloud	Cloud	$\frac{1}{16}$	*	$\frac{1}{16}$

N.B.—“Cloud” means no precipitate. “Trace” means a deposit not covering bottom of test-tube.

* means no determination.

A STUDY
OF THE SO-CALLED
TENDON-REFLEX PHENOMENA.

BY

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THE following paper records a series of observations on the two forms of muscular spasm known under the above designation (described first by Erb and Westphal), and which have been associated with spinal sclerosis: (1), the movement which occurs under normal conditions at the knee-joint when the patellar tendon is struck, the "patellar-tendon reflex" of Erb, the "knee phenomenon" of Westphal, which is absent in most cases of locomotor ataxy, and, (2), the movement which occurs at the ankle-joint in certain forms of disease of the spinal cord (probably lateral sclerosis) when the extensor muscle and tendon are suddenly put upon the stretch—the "Achilles-tendon reflex" of Erb, the "foot phenomenon" of Westphal.

Although the characters of each of these movements, and the mode of their production, have been made familiar to the profession in this country by the description of them by Dr. Grainger Stewart,¹ and Dr. Buzzard,² it is necessary, for the sake of clearness, to state briefly the method by which they may be obtained.

¹ "Clinical Lecture on Tendon-Reflex," 'Medical Times and Gazette,' 1878, January.

² "Lectures on Sclerosis of the Cord," 'Lancet,' 1878, July.

KNEE-REFLEX.

The knee-reflex, "patellar-tendon reflex," or "knee-phenomenon," is the contraction which may, under normal conditions, be excited in the quadriceps extensor of the knee-joint, by striking the patellar tendon a light but sharp blow, the tendon and muscles being previously put gently on the stretch. Any position will suffice which involves tension in this tendon, i.e., any flexion of the knee-joint, but as the effect of the resulting contraction can be best observed by the consequent movement of the leg, a position is most convenient which leaves the leg free to move, such as sitting with the legs hanging freely, or with the leg to be tested placed across the other, or across the observer's arm, so that the knee-joint rests in a position of flexion at an angle a little greater than a right angle. This is especially convenient, because it prevents the blow on the tendon from causing an increased flexion of the leg, the swing back from which may, if the leg is hanging vertically, be mistaken for the reflex contraction.

Schültze and Fürbringer have shown that it can be obtained with great readiness by a blow on the exposed tendon of an animal. It may also sometimes be obtained by a blow upon the rectus tendon above the patella, by a blow on the muscular fibres of the rectus itself, and by an oblique downward blow on the patella, as readily as by a blow upon the patellar tendon. Each blow is followed by a single contraction, but as long as the blows are continued the contractions follow, and by making the blows succeed one another with sufficient rapidity, the contractions occur so closely that only slight relaxation takes place in the intervals, and thus a state of almost tetanic contraction is produced. The blow, if sharp, causes a peculiar sensation in the part struck, and sometimes also in the muscles of the thigh. Under pathological conditions the sensation may amount to actual pain, although the tap is light.

In order to ascertain what are the variations in this phenomenon under normal and abnormal conditions, I have,

with the kind assistance of Mr. A. E. Broster, resident medical officer at the National Hospital for the Paralysed and Epileptic, examined 300 individuals, of whom 150 were suffering from no known paralytic condition of the lower extremities. Of these some were, as far as was known, in perfectly normal health, but the majority were the subjects of simple epilepsy. The hand only was employed, the fingers being extended and the patellar tendon struck with the edge of the little finger.

Of the whole 300 cases the movement was entirely absent on this mode of testing, on both sides, in seventeen. It is possible that the use of the percussion hammer, as proposed by Westphal, might have elicited a slight reflex in some of these cases. The observations were, however, carefully made, and the results are, I think, accurate as regards the mode of testing employed. Four of the seventeen were suffering from undoubted locomotor ataxy, a fifth had atrophy of the optic nerves, and commencing ataxy was suspected. Of the remaining twelve, two were under treatment for paraplegic weakness of legs, but presented no indication of ataxy. These were :

(1.) A man, æt. 52.

(2.) A man, æt. 45; with some weakness of the legs, especially in the flexors, which had apparently been left by an attack of spinal meningitis. Sensation of the legs was normal.

The remaining ten patients were as follows :

(1.) A woman, æt. 58, who had had an attack of right hemiplegia, but had recovered so as to be able to walk ten miles. The patellar reflex was absent on both sides (in all the cases now under consideration).

(2.) A man, æt. 58, suffering from some mental disturbance and slight weakness, deafness, and considerable auditory nerve vertigo.

(3.) A man, æt. 36, suffering from tumor cerebri, syphilitic, of which he afterwards died. When tested he was suffering from slight weakness in the right arm, but was able to walk a long distance.

- (4.) A girl, æt. 11, suffering from chorea, a good walker.
- (5.) A man, æt. 19, suffering from epilepsy, whose legs were curved from old rickets, and who was not able to walk more than a mile.
- (6.) A man, æt. 30, suffering from epilepsy and considerable cardiac disease; able to walk about two miles.
- The remaining four cases were all young epileptics.
- (7.) A boy, æt. 7; a bad walker.
- (8.) A girl, æt. 19; a bad walker.
- (9.) A girl, æt. 22; walking power not good, about a mile.
- (10.) A girl, æt. 20; walking power not good.

Whenever the reflex was apparently absent it was tested repeatedly with great care, without the intervention of the clothes or with only a single garment.

It would thus appear that the knee reflex cannot be obtained, at least with the hand, in about four per cent. of patients suffering from disease of the nervous system who present no symptom of locomotor ataxy, and that in many of these, from sex and age, the possibility of commencing ataxy can be certainly excluded; but that, in most of them, the loss is associated with defective power of continued exertion with the legs.

In at least half the cases of nervous disease in which the knee reflex cannot be obtained with the hand the patient is not suffering from locomotor ataxy. To those who have followed recent discussion on this subject this statement will appear surprising. Hitherto, apparently, the symptom has rarely been searched for except in ataxics or in a few persons in normal condition for the sake of comparison.¹ Before considering the conditions in which it is commonly absent, the facts collected suggest several points of interest regarding the phenomenon.

¹ Since this paper was read Dr. Berger has published ('Centralbl. für Nervenheilkunde,' No. 4, 1879) the results of the examination of 1409 healthy individuals. He found that no reflex could be obtained by any method of examination in 22, or 1.56 per cent. In comparing this result with those which I have obtained, it must be remembered that most of the persons tested by me were suffering from some affection of the nervous system,

In three cases only was the reflex absent in one leg, and present, although slight, in the other. One was a woman aged 47 who had had an attack of right hemiplegia, from which she had recovered sufficiently to be able to walk several miles. The reflex was present, although slight, in the leg which had been paralysed, and was absent in the other. The second case was a woman aged 41 who had suffered in early life from chorea and subsequently from epilepsy. She had some weakness of the legs, greatest in the left, and was able to walk only one mile; the knee reflex was present though slight in the weaker leg and was absent in the other. In the third case, it was found that, on the side on which the reflex could not be obtained, there had been an old transverse fracture of the patella, and the two halves were some distance apart.

Twenty-seven cases of hemiplegia from cerebral disease were examined. They were of various duration and degree of recovery: most attended as out patients and so were able to walk. The reflex was equal in the two legs in 13 of the cases, and it was unequal in 14. In the latter, without an exception, the excess of movement was on the paralysed side. The occurrence of this excess seems to bear no necessary relation to the development of the ankle clonus, which is often present in old hemiplegics. This ankle clonus was marked in several of the cases in which the knee reflex was equal on the two sides and it was absent in several cases in which the excess of movement on the paralysed side was very large.

In order to estimate the average degree of movement, apart from any weakness of the legs, the range of movement (of the foot) which could be excited by such a tap as usually develops the maximum effect, has been compared in one hundred epileptics whose walking power was reasonably good. The amount of movement was estimated by the eye, checked by an occasional measurement. Of the 100, 37 were women and 63 were men. The results are shown in the following table:

Movement in inches.	Men.	Women.	Total each degree.
$\frac{1}{2}$...	3 ...	1 ...	4
1 ...	22 ...	6 ...	28
2 ...	19 ...	18 ...	37
3 ...	14 ...	11 ...	25
4 ...	3 ...	1 ...	4
5 ...	2 ...	— ...	2

It appears from this that in 90 per cent. of individuals possessing good walking power the range of movement is between 1 and 3 inches. The mean of the whole is just 2 inches, and about as many present a slight excess above the average movement, as present a deficiency. The reflex was entirely absent in no patient with good walking power, although in 4 it was slight, not more than half an inch. In only 6 did it exceed 3 inches.

The sexes present some differences, if it be permissible to generalise from so limited a number of cases. A larger number of men present a considerable excess and a considerable deficiency. Two fifths of the men had a reflex of under 2 inches, and only one fifth of the women. The average for men is a little below 2 inches, for women a little above 2 inches.

Much discussion has been given to the question whether the contraction is, as maintained by Erb, a true spinal reflex, or whether it is a simple effect of the blow on the tendon upon the fibres of the muscle, causing their sudden contraction, as originally maintained by Westphal. That it is a true reflex action is commonly considered to be proved by experiment on animals, since it ceases on section of the anterior crural nerve. Still stronger evidence is afforded by the fact that it is commonly absent in a disease, locomotor ataxy, in which the spinal cord is diseased, but in which the state of motor nerve and muscle is perfectly normal.

The graphic study of the movement is in entire accord with the theory of its reflex character. I show a series of tracings (Fig. 1) taken with a revolving drum, the writing pen being attached to the foot, which demonstrate that the muscular contraction occurs at such an interval after the

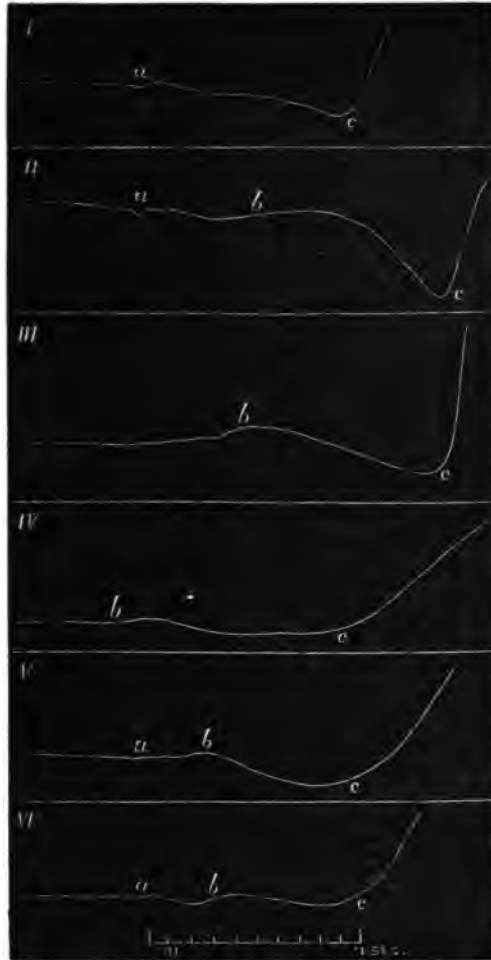
stimulating tap, as, according to physiological data, would be needed for the processes involved in the necessary spinal reflex action, *i.e.*, (1) the conduction of the afferent impulse to the cord, and of the efferent impulse from the cord to the muscle; (2) the process of reflex action in the cord itself; (3) the period of latent stimulation in the muscle.

(1) The length of nerve fibre to be traversed from the tendon to the cord and back again to the middle of the rectus muscle is about a meter and a half. The rate of transmission of nerve impulses in man is about 33 meters per second. For this, therefore, $\cdot 045$ second would be needed; (2) the shortest time needed for a simple reflex process in the centre, has been estimated at $\cdot 05$ second.¹ (3) The period of latent stimulation of the muscle may be taken as about the $\cdot 01$ second. Adding together these periods we have a total of $\cdot 045 + \cdot 05 + \cdot 01 = \cdot 105$, or a little over the $\frac{1}{10}$ ths, *i.e.*, $\frac{1}{10}$ th of a second. I have found that the interval between the tap on the tendon (*a*, fig. 1) and the movement of the foot (*c*) which indicates the commencement of the contraction, in two normal individuals, varied somewhat, but that the shortest interval is the $\cdot 09$ second, and the longest $\cdot 15$ second (fig. 1, Nos. I—IV). Generally it is the $\cdot 10$ or $\cdot 11$ second. In a case of lateral sclerosis, tracings from which are shown in the same figure, Nos. 5 and 6, the interval was always the $\cdot 10$ second. This correspondence between the period found to elapse and that estimated to be necessary is remarkably close, and accords perfectly with the view that the phenomenon is a spinal reflex.

But some of the tracings show another, and very interesting character; the interval between the stimulation and the movement is occupied by a slight rise (*b*) and by a fall, often more considerable than the rise. This may, at first sight, be regarded as the mechanical effect of the blow upon the leg; but a comparison of several tracings shows that this is not its explanation, for the rise is often preceded by an interval, after the blow, during which no movement occurs, an interval not exceeding $\cdot 05$ second. This rise and fall

¹ Exner, 'Pflüger's Archiv,' VIII, p. 526.

FIG. 1.



Tracings of knee-reflex. I—IV, tracings from two healthy individuals; V and VI, from a case of disease of the spinal cord (lateral sclerosis?); *a*, tap upon patellar tendon (not indicated in III and IV); *b*, slight (direct?) muscular contraction; *c*, commencement of reflex contraction. The tracings are all taken at the same rate; the divisions on the scale correspond to hundredths of a second.

must then be due to a slight contraction, and subsequent relaxation of the muscle. The interval is not enough for the occurrence of a spinal reflex action, and there can be little doubt that we have here the direct effect upon the muscular fibres of the blow on the tendon. The sudden slight tension upon the fibres causes a brief slight contraction, succeeded by relaxation, and followed by the strong contraction which results from the spinal reflex. Thus, although the visible movement is not the result of the mechanism to which Westphal first attributed it, that mechanism is in operation and produces an effect, not, however, appreciable by ordinary methods of observation.

I am aware that some measurements which have recently been made by Burckhardt¹ and Tschirjew,² have assigned to the interval a much shorter duration than my own observations assign—not more than .04 or .05 second. But the method of the experiments—by arranging an apparatus so that the swelling of the muscle broke an electric circuit during the rigid fixation of the foot—seems to me to be open to many fallacies from which the simpler method I have adopted is free. In spite of the brief duration they assign, both observers conclude that the phenomenon is reflex, although they admit that the time they assign is actually insufficient for the mere passage of the impulses, to and fro, along the nerves, and leaves no time at all for the reflex process in the cord. The occurrence, under some circumstances at least, of a slight muscular contraction before the more vigorous one, accounts, perhaps, for the discrepancy between these results and those I have obtained.

It should be mentioned that Burckhardt states that, in animals, the phenomenon, although arrested by section of the crural nerve, persists after division of the spinal roots and hence concludes that it is a reflex from the ganglia on the posterior roots. But this is opposed to the results of Tschirjew who found it at once arrested by section of the posterior roots

¹ G. Burckhardt, 'Ueber Sehnenreflexe,' Bern, 1877. 'Centralblatt. für die Med. Wissensch.,' 1878, 401.

² 'Archiv für Psychiatrie,' Bd. viii, 1878, No. 3.

of the sixth lumbar pair, and cannot, I think, be allowed much weight in the face of the pathological evidence of the relation of the reflex to changes in the spinal cord.

In animals it has been observed that the muscular contraction excited by the tap on the exposed tendon, sometimes occurs not only in the leg struck but also in the corresponding muscle of the other leg. I have several times observed the same thing in man, in cases in which the reflex movement is considerable, especially in cases of old hemiplegia. In some of these a simultaneous contraction in the muscles of the back was also produced, jerking the body and even the head backwards. I need not point out what strong, even conclusive evidence this extension of the reflex affords, for believing the seat of the reflex process to be in the spinal cord itself.

The stimulus which excites the reflex contraction is commonly regarded as originating from the part struck, the tendon. Tschirjew, however, asserts that it persists after division of all the nerves from the tendon. It is certainly possible that the sudden effect of the blow on the tendon upon the fibres of the quadriceps may originate the afferent impulse, but the peculiar sensation in the tendon when struck suggests that, in this case, the afferent impulse does originate in the tendon. It is probable also that the afferent impulse may originate from several localities; since an oblique downward blow on any part of the patella, a blow on the tendon above the patella, or on the muscle, will suffice to produce it.

The visible phenomenon being thus those of a spinal reflex, the integrity of the path to and from the cord, and of the reflex mechanism in the grey matter of the cord, will be necessary for its production; and damage in any one of these situations, to the sensory fibres, the motor fibres, or the grey matter, may be expected to prevent its occurrence. Facts are in perfect accord with this conclusion.

(1.) The phenomenon is absent in most cases of locomotor ataxy in which, as is well known, the region of the cord damaged is that, and may be that only, through which the

fibres of the posterior nerve roots pass, viz.—the outer portions of the posterior columns ; and the absence of the reflex in this disease, is often related in a remarkable way to the degree of damage to the posterior nerve roots, as evidenced by the symptoms.

(2.) Damage to the motor nerve roots must, of necessity, interfere with the reflex ; but I am not aware that any instances of its loss have yet been recorded in which the lesion was limited to this situation. In the experiments of Fürbringer the crural nerve was divided, which contains, of course, the sensory as well as the motor fibres involved.

In one case, under my own care, in which the reflex is entirely lost there is probably pressure on the nerves by meningitis ; the recti, however, are completely paralysed and wasted. Sensation is normal.

(3.) Damage to that part of the anterior grey cornu of the cord, to which the quadriceps extensor is related, prevents absolutely the production of the reflex. It is usually lost—always as far as my experience has hitherto gone—in cases of atrophy of the lower extremities in which the quadriceps extensor is in any considerable degree involved, and often when its affection is slight, and this in cases in which there is not the slightest impairment of sensation. This fact has also been pointed out by Westphal. I have found it completely absent even in cases in which the quadriceps extensor retained sufficient power to maintain the complete extension of the knee when the leg was raised into the air. The reflex is lost, not only in ordinary cases of atrophy, but also in pseudo-hypertrophic paralysis, invariably in the later stages of the disease, when the quadriceps extensor is always considerably damaged.

In the diseases in which the knee reflex commonly disappears, the central change varies in distribution and exact seat in different cases, and it is not surprising, therefore, that we meet with exceptions to the rule of its absence. It is probable that instances will be met with in which it is present, under some of the conditions in which it is commonly lost, in each of the several forms of lesion. I will, however,

confine myself to some remarks on its occasional presence in the disease in which its absence has attracted most attention, viz. locomotor ataxy.

Several observers have alluded to persistence of the knee reflex in well-marked cases of locomotor ataxy. I have met with two such cases, which illustrate also the conditions to which this preservation of the knee reflex appears to be especially related, namely, the absence of evidence of damage to the posterior nerve roots. The patients were under my care at the National Hospital for the Paralysed and Epileptic. In two other cases of ataxy, in which the knee reflex was preserved, it is probable that there was combined lateral and posterior sclerosis of the cord.

CASE 1. Well-marked locomotor ataxy, of gradual onset, without any recognisable affection of sensation, without lightning pains, and with well-marked knee reflex.

William L—, æt. 43. Difficulty in walking commenced gradually at thirty-eight (in 1873), and increased so that in six months time he could not walk although he could move his legs freely as he lay in bed. Rest and a change to the sea side restored his walking power.

When seen in June, 1878, he walked unsteadily, in the most characteristic ataxic manner, raising his legs too high. He could not stand with his feet near together, and he fell down when he closed his eyes. There was no affection of his arms, ocular movements, sight, or pupils. Sensation on the legs and soles was normal to both touch, pain, and heat. The only point in which there was any doubt was as to whether he could quite accurately localise a painful impression upon one great toe. On each side there was a very well marked patellar tendon-reflex, which still (November, 1878) persists in the same degree.

CASE 2.—Symptoms of locomotor ataxy; knee-reflex present; lightning pains on standing only; no affection of sensation.

Wm. H. D—, æt. 54. At fifty-two years of age found that a small quantity of beer made him very unsteady, and soon afterwards that the unsteadiness was constant, irrespective of beer. For a year and a half the unsteadiness, slowly increasing, remained the only symptom. He then began to suffer from severe "gnawing"

pains in both legs, and subsequently from sharp momentary pains on movement, passing from the sacral region down the back of the thighs as far as the knees, "like a flash of lightning and gone again." They did not occur when he was at rest. When he came under treatment the muscular power in the legs appeared to be unimpaired, but the ataxic gait was well marked. He walked in an uncertain manner, often raising his feet too high; on turning round suddenly he almost fell. Closure of the eyes rendered standing almost, and walking quite, impossible. The knee reflex was considerable in each leg, the movement of the foot being about four inches. Not the slightest ankle clonus could be obtained. Sensation in the legs and feet, repeatedly tested by myself and others, was perfectly normal to touch, pain, and heat. The muscular sense also appeared to be normal. (Since that time his ataxic gait has increased. The gnawing pains continue, and the lightning pains occur when he is standing still, as well as when he is moving, from the hip down the back of one or the other leg. The knee reflex continues excessive, and sensation is still perfect, June, 1879.)

CASE 3.—*Ataxy and weakness of legs; lightning pains in both legs, but more in one than in the other, the knee reflex lost in the leg in which the lightning pains were most intense, present in the other.*

James F—, æt. 38. No history of syphilis. At thirty-three began to suffer from weakness and pains in the legs, felt in both, but far more severe in the left leg than in the right—sharp darting pains, coming for a moment and then going away, and in the left leg accompanied with a sudden involuntary movement of the leg. The pains in the right leg were of precisely the same character, but less severe. The weakness came on gradually at the same time, first, and always worse in the left leg. When seen there was a considerable amount of loss of power in the legs, more marked in the flexor muscles than in the extensor, and on the whole less in the left than in the right leg. He could stand alone on the right leg for a moment, not on the left. Over and above the weakness there was considerable unsteadiness of movement, and he was obliged to stand with his feet far apart, just as an ordinary ataxic. The electric irritability of the muscles was unchanged; there was no wasting. Sensation to touch in left leg was normal above the knee, diminished below, and lost on the sole. On the right leg it was lessened below the knee, although not so much as in the left leg, and

on the right sole a touch could just be felt. Pain was felt as such on each leg, but not at all on the left sole, moderately on the right sole. Heat was not perceived as such on either leg below the knee. The patellar reflex in the right leg was excessive, the movement of the foot being about four inches; in the left leg it was entirely absent. On faradising the skin outside the knee-joint (which in paraplegics often causes a strong reflex contraction in the flexors of the hip) the right hip was strongly flexed, the left not at all. I have no note of the existence of ankle reflex.

This may, I think, be regarded as probably a case of combined lateral and posterior sclerosis, with much greater damage to the posterior nerve roots on the left side than on the right.

The following case is probably also of the same character:

CASE 4.—James L—, æt. 27; never quite well since rheumatic fever, at 25. Three months before being first seen he found that he could not walk well. When seen he could only walk a mile with difficulty. He stood unsteadily, with the legs far apart, but could just balance himself with the feet close together and his eyes shut. His gait was unsteady; the feet brought down too suddenly, and the unsteadiness was increased by closure of the eyes. The walk was precisely like that of a moderate degree of ataxy. Sensibility in the legs and feet to heat, touch, and pain was perfect. The knee reflex was excessive in each leg. Ankle clonus slight, but "front-tap contraction" (see p. 292) very marked. No sharp pains, but dull, "numb-like" pains in both legs.

In another case which has come under my notice, a woman, with symptoms of spinal sclerosis, weakness of legs, with unsteadiness of gait in excess of the weakness, and slight impairment of sensation, the knee reflex was present when she was first examined, but, three months later, had entirely disappeared. I had not an opportunity of ascertaining whether its disappearance coincided with any increase in the loss of sensation.

These cases show, I think, that the preservation of the reflex in simple locomotor ataxy coincides with such a slight degree of damage to the fibres of the posterior nerve roots as does

not involve appreciable loss of sensation. It does not follow that the fibres which convey sensation from the limb are the same as those which convey the impression from the tendon. This latter may have a somewhat different course, and suffer when the damage is extensive, and escape in some cases in which it is slight, while other cases, in which the loss of the reflex has preceded other symptoms of ataxy, seem to suggest that the position of the fibres from the tendon is one in which the posterior sclerosis is apt to commence.

Another indication of damage to the posterior nerve roots is the occurrence of lightning pains which, as Westphal and Buzzard have shown, commonly coincides with absent reflex. But the second and third cases I have recorded show that, in some conditions, the reflex may persist after lightning pains have occurred for some time. In the second case, however, they only occurred when the patient was standing or moving. In Case 3 the lightning pains, although present in both legs, and occurring at all times, had been most intense in the leg in which the patellar tendon reflex was lost.

ANKLE CLONUS.

The ankle clonus, or ankle reflex, is the clonic spasm which can be obtained at the ankle-joint in certain cases of weakness of the legs. To obtain it, it is necessary merely to make a sudden dorsal flexion of the foot. It is obtained most readily when the knee-joint is nearly but not quite extended. If, then, the hand be placed against the ball of the foot, and the ankle-joint flexed by a sudden pressure of the hand, so as to make the Achilles tendon tense, there follows, apparently instantly, a brief contraction of the gastrocnemius, sufficient to move the foot and hand a little in the direction of extension, and this ceasing, the pressure of the hand being kept up, another similar contraction follows, and so on as long as the pressure of the hand is maintained. When the hand is removed, the contractions cease, immediately, or after two or three weaker contractions. If the foot be passively extended, so as to relax

completely the Achilles tendon, the contractions instantly cease.

It is not necessary that the passive flexion should be made by pressure of the hand on the sole. The clonus may be produced equally well by grasping the extremity of the os calcis and pulling the heel down.

If the passive flexion be made gently and gradually a considerable tension may be put on the tendon without exciting the movement. If, however, the Achilles tendon or the gastrocnemius muscle be then tapped, a contraction instantly occurs, followed by others, the pressure of the hand being maintained.

The phenomenon occurs commonly in those diseases of the spinal cord in which there is reason to believe that there is sclerosis of the lateral white columns, either primary or secondary to disease of the cord higher up, or secondary to a lesion in the brain. It is thus frequently met with in both hemiplegia and paraplegia. Its occurrence in both legs in cases of hemiplegia has been noted, and I have confirmed this observation, having found it in one case even more marked in the sound leg than in that on the hemiplegic side.

With a revolving drum, I have taken tracings of this movement in ten cases, the number of tracings being in all about forty. The writing lever was fixed to the side of the foot. These tracings show certain characters which deserve description.

1. The regularity of the movement, apparent to simple observation, is still more strikingly seen in its record (Fig. 2). The series of curves traced are almost as regular as those of a tuning fork. They are, however, liable to slight variations, both in different individuals and in the same individual at different times.

The average number of contractions per second is nearly six, the variations being, in the whole series of cases, between five and seven per second. This applies to the frequency of the contractions when they are set up. The first one or two may be at a longer interval. The average duration of each movement, and the interval between them from the

commencement of one contraction of the gastrocnemius to the commencement of the next, is sixteen hundredths ($\cdot 16$) of a second, the shortest being thirteen ($\cdot 13$), and the longest twenty hundredths ($\cdot 20$) of a second. The average length may thus be roughly put at one sixth of a second. The facts from which these estimates are made are contained in the following table.

Measurement of the Frequency of Movement in the cases of Ankle Clonus.

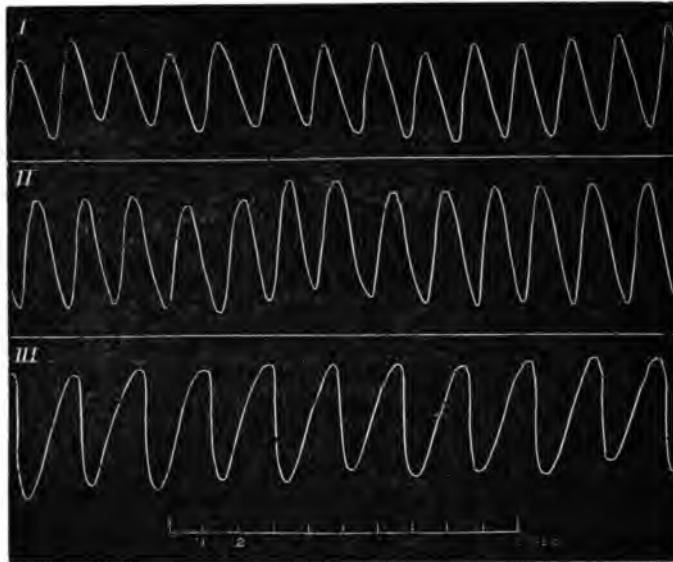
Case.	Disease.	Number per second.		
		Minimum.	Maximum.	Average.
1	Primary (lateral ?) sclerosis . . .	6	7.3	6.8
2	" " " " . . .	6.5	8	6.8
3	" " " " . . .	5	5.8	5.4
4	" " " " . . .	5.1	6.3	5.7
5	Descending sclerosis ; caries of spine . . .	6.3	7.5	6.9
6	" syphilitic growth in cord . . .	6.15	6.3	6.2
7	" hemiplegia . . .	7	7	7
8	" " " " . . .	5.2	5.8	5.3
9	" ? " " . . .	5	5.8	5.4
	Average	6.17

It is not easy to adjust the apparatus to the foot so as to obtain the regular curve which the lever actually describes, but from an examination of the most successful tracings it appears that the rise of the lever is more sudden than its fall, but the curve at the top renders the commencement of the fall very nearly midway between the commencement of two successive contractions ; one contraction ceasing on an average about one tenth of a second before the commencement of the next.

The actual amount of muscular contraction, i.e., the extent of movement, varies in different cases. It was several times estimated by making the distance of the writing pen from the malleolus (corresponding to the fulcrum) a multiple of the distance from the insertion of the tendo Achillis to the malleolus. The contraction was found to vary from 1.5

millimeter to 3 millimeters, giving a movement at the tip of the great toe of 1 to 2 centimeters.

FIG. 2.

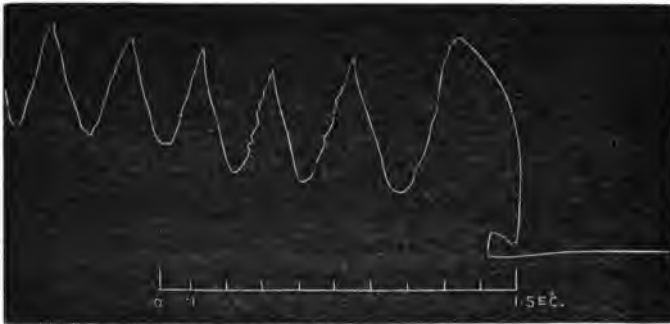


Tracings of the ankle clonus. I, primary spinal (? lateral) sclerosis; II and III, lateral sclerosis after hemiplegia. The tracings read from right to left. The divisions of the scale are tenths of a second.

During the intervals of contraction, the relaxation of the muscle is not complete. This is shown by the following tracing (fig. 3) which represents the commencement of the contraction started by a tap upon the tense tendon. The straight line was traced by the writing lever during the maintenance of the tension by gentle pressure. The tap caused a slight rise and backward movement of the lever, followed by a sudden strong contraction of the muscle, shown by the ascent, first vertical and then curved. The descent which follows the cessation of contraction does not fall so low as the line from which the lever started, the descent (relaxation) being interrupted by another rise (con-

traction). It is to be noted, further, that the line joining the lowest portions of the curves rises for a time, indicating that the degree of residual contraction increases. In this case the degree of residual contraction was at first 1.5 mm. and gradually increased to 3 mm.

FIG. 3.



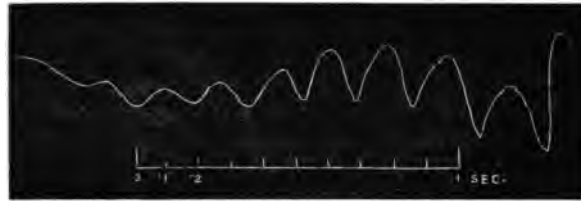
Ankle clonus set up by a tap on the tendo Achillis during passive dorsal flexion of the foot. The initial contraction of the gastrocnemius is seen to occur almost instantly, to be greater in degree and in duration than the contractions which succeed. The lowest subsequent descent does not fall so low as the line indicating the position of the foot before the tap, *i.e.* the relaxation is incomplete. The tracing reads from right to left. The divisions on the scale indicate tenths of a second.

This peculiar clonic spasm, which is so common in the gastrocnemius has not, I believe, been described in any other muscle. On several occasions, however, when it has been well-marked in the calf, I have succeeded in obtaining it with almost equal readiness in the plantar muscles of the great toe, the abductor and flexor brevis pollicis. These muscles are inserted into the base of the first phalanx of the great toe, and to obtain the movement it is only necessary to fix the foot with one hand in a position of extension (*i.e.* plantar flexion) so as to prevent the occurrence of the gastrocnemius reflex, which would obscure the other, and then to make a sudden passive extension of the first phalanx of the toe. The resulting movement is quite similar in

its characters to that of the ankle-joint. This is shown by the tracing of it (fig. 4) which I have obtained by fastening the writing lever to the great toe only.

In most cases the movement ceases after five or six contractions as it does also, in slight cases, at the ankle-joint. At other times, and by careful manipulation, a long series of contractions may be obtained. One of the tracings shows a series of eighteen. The time of this toe clonus is almost the same as that of the ankle clonus. One tracing (not figured) shows the identity of time in the toe clonus and in the ankle clonus which was taken at the same time, from the same patient, a girl suffering from a primary (lateral ?) sclerosis of the cord. The frequency of the toe clonus in this case was,

FIG. 4.



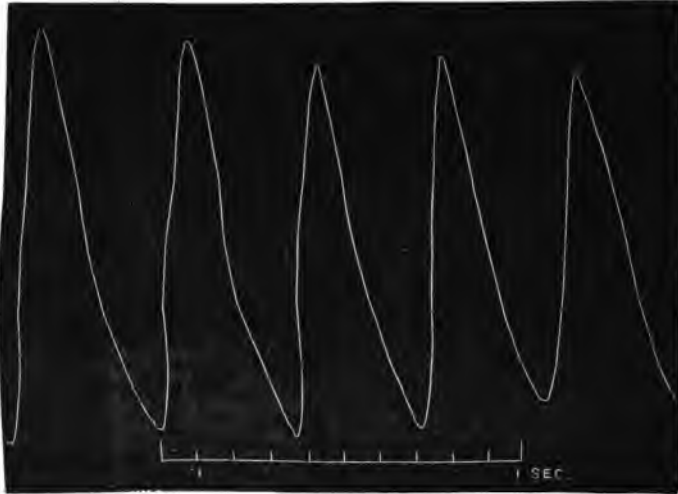
Toe clonus; contractions in abductor pollicis on passive extension of great toe in a case of spinal (lateral ?) sclerosis. The tracing reads from right to left. The divisions on the scale indicate tenths of a second.

maximum 6.15, minimum 6, mean 6.1. The mean of the ankle clonus being 6.8 contractions per second.

In several cases presenting the symptoms usually ascribed to lateral sclerosis of the cord, I have succeeded in obtaining a lateral ankle clonus by grasping the foot with the hand and pressing it laterally. In most of these cases the clonic spasm was evidently in, and confined to, the peronei, and could only be obtained when the foot was pressed inwards. In one case, however, it could be obtained in either the peronei or the tibialis posticus according as the foot was pressed inwards or outwards. The time appeared to be the same as that of the ordinary ankle clonus, and of the toe clonus,

In two cases in which the patellar tendon reflex was very marked, the contraction produced in the extensor of the knee was very often followed by others, in uniform succession, continuing for a considerable time; a knee clonus, resembling the ankle clonus in regularity. It differed from the ankle clonus, however, in the much longer interval between each contraction. In one case tracings were taken, and fig. 5 shows the characters of this movement, only two and a half contractions occurring per second. In each case it had the same slow time. Its probable significance and origin will be considered presently.

FIG. 5.



Knee-clonus — clonic contractions in the quadriceps extensor femoris in a case of advanced disease of the spinal cord (spastic paraplegia). To be read from right to left. The extent of movement gradually increasing; its frequency is seen to be about half that of the ankle clonus.

To return to the ordinary ankle clonus.

Two theories have been proposed regarding the nature of this phenomenon, analogous to those which have been suggested in explanation of the knee reflex. The one, that of Erb, regards each contraction as a reflex from the spinal

cord, the excitant being the stimulation of nerves in the tendo Achillis by the sudden tension, alone or combined with a tap which may act by increasing the tension previously gently applied. The stimulation is constantly repeated by the maintained passive flexion, whenever the muscle relaxes. On the other hand Westphal has suggested that the tension must affect directly the muscular fibres of the extensors of the ankle, in which the contraction occurs, and that this excites them to immediate contraction, so that the phenomenon is not really reflex. A third explanation may however, be proposed viz., that the phenomenon is really reflex but that the stimulus originates, not in the tendon but in the muscle, by the irritation of its afferent nerves by the sudden tension on its muscular fibres. There are thus two points to be considered, first, is the spasm produced by a stimulation of the tendon or of the muscle? and secondly, if the latter, is the effect direct or reflex?

In the first place the spasm cannot be excited by any stimulus applied to the tendon which does not increase the tension. It may perhaps be doubted whether this is true of the gentle tap which, when the tendon is made tense, excites the contraction of the muscle. But that even this gentle tap produces its effect by causing an increase of tension is shown by the following fact. If the tendon be gently made tense, in the manner already described, a tap on the side of the tendon will produce the muscular contraction almost as readily as one upon the posterior aspect. But if the movement of the tendon be prevented by placing the tips of two or three fingers against the opposite edge, the tap produces no effect. Take the fingers away, and the same tap at once causes the contraction of the muscle. Moreover, no other means of stimulation of the tendon, if they do not increase its tension, will excite the spasm. The tendon is sensitive to a pinch, as anyone may ascertain by making the experiment on himself; but pinching the tense tendon does not excite the contraction. This is the more worthy of special notice, because, in the patients who present the ankle clonus, a pinch of the tendon does commonly excite a very marked reflex

spasm, but this is similar in character to that which results from a cutaneous stimulation, and in it the flexors predominate. This will be considered more fully presently. But any stimulus which increases the tension in the tendon, must increase the tension in the muscle also; and thus no stimulus is effective, applied to the tendon, which does not act also upon the muscle. No fact can be adduced in favour of the view that the stimulus originates in the tendon which is not equally in favour of its origin in the muscle.

But when gentle passive dorsal flexion is made, a tap upon the muscle will excite the contraction as readily as a tap upon the tendon. Now a tap on the muscle will cause a much slighter increase in the tension of the tendon than a tap on the tendon itself; and so this fact is in favour of the view that the stimulus originates in the muscle. But it is not conclusive, for it cannot be denied that the tap may increase, although very slightly, the tension in the tendon.

Additional light is, however, thrown on this question by an examination of the second point. What evidence is there regarding the reflex nature of the phenomenon? The first fact is that the clonus (obtained in this manner) occurs only in cases in which there is the strongest reason to believe that there is an organic change in the cord, and a change which causes an exaltation of the knee reflex, which, as we have seen, must be regarded as of spinal origin. But this fact, the constant relation of the ankle clonus to spinal disease, is not evidence of its spinal origin, because it is quite conceivable that disease of the cord, by the withdrawal or the exaltation of a central influence, may leave the muscle in a condition to respond, in an abnormal manner, to direct stimuli. We see the muscular fibres in progressive muscular atrophy quiver unduly when struck, but we do not regard their quivering as therefore a spinal reflex. Other evidence on the question has therefore to be sought.

Does any stimulus applied at a distance from the muscle and tendon excite the peculiar spasm? I have found one stimulus which does, at first sight, appear to fulfil this condition, and which is in itself a phenomenon of considerable

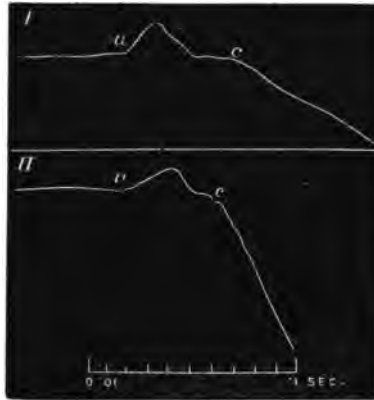
interest and practical importance. If, when the ankle is gently flexed in the manner described, so that no spasm occurs, the tibialis anticus or adjacent muscle be gently tapped, there occurs, not a contraction in the muscle tapped, but a contraction in the gastrocnemius, causing a slight extension movement at the ankle, not followed by others, but a single isolated contraction. That this is not the result of any increased tension of the tendon, such as might result from the tap tending to increase the flexion of the ankle-joint, is shown by the circumstance that a similar and even stronger tap upon the adjacent tibia produces usually no effect.* This phenomenon, which may be termed the "front-tap contraction" may generally be obtained in the cases in which the ankle clonus is present.

Here then we have a contraction in the calf muscles similar to that which initiates the clonus, and produced under similar conditions, but by a stimulus applied at a distance from muscle and tendon. It appears at first sight to be an unmistakable reflex action. Regarding it as such, I took, to ascertain its characters, several tracings by the same means which the other tracings were taken, but with a rapid movement of the drum so as to measure accurately the interval between the stimulus and the contraction. Two of these tracings are now shown (fig. 6). The tap, it will be seen, is very distinctly indicated in the brief rise of the lever (*a*), and the contraction of the gastrocnemius by the fall (*c*), which commences a little time after the movement from the tap has ceased. It will be seen, however, that the interval from the commencement of the tap to the contraction, is, in the one case, only .04 and in the other .05 second, while the interval between the middle of the rise occasioned by the tap is only .03 and .04 second respectively. This is, according to received facts, insufficient for the occurrence of a spinal reflex. The mere passage of the impulse to and from the cord would occupy a longer time, about the .06 of a second, and as before stated, physiological facts suggest that nearly as much

* In rare cases, in which the irritability is very great, a tap on the tibia will produce the contraction.

time (.05 sec.) would be needed for the simplest reflex process in the centre. The corresponding interval in the case of the knee reflex, in which there is a shorter distance of nerve fibre to be traversed, has been seen to occupy double the period in question.

FIG. 6.



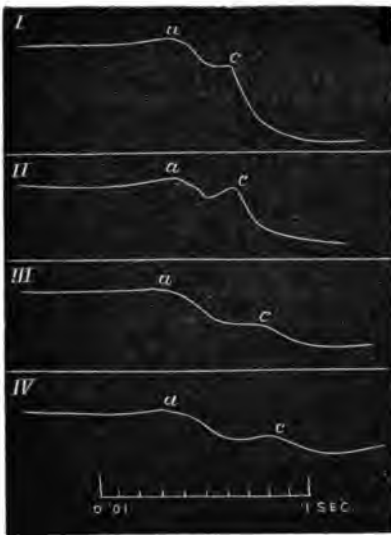
Tracings of front-tap contraction, *i.e.* the contraction in the gastrocnemius excited by a tap on the tibialis anticus during passive dorsal flexion (lateral flexion?). The interval between the commencement of the rise (*a*), indicating the tap, and the descent (*c*), due to the contraction of the gastrocnemius, is in the upper tracing .05, and in the lower one .04 second. The divisions of the scale indicate hundredths of a second.

This fact seems entirely to dispose of the hypothesis that the contraction can be a reflex from the spinal cord. If not a reflex, how does the tap produce it? If the fingers of the other hand be placed upon the middle of the calf, when the tap is made, during the dorsal flexion of the foot, a distinct vibration will be perceived, when the tap is upon the muscles in front of the leg, which is absent when the tap is made upon the tibia. This vibration must be transmitted through the muscles and the intermuscular septum; and it seems probable that this vibration may so disturb the equilibrium of the tense fibres of the gastrocnemius and soleus as to excite them to contraction, by direct stimulation.

I have also measured, in two cases of spastic paraplegia the interval between a tap on the gastrocnemius itself and the resulting contraction of the muscle, and also the interval between the tap on the Achilles tendon, and the contraction it excites. If the passive flexion of the foot and tap are gentle, each of these stimuli will excite a single contraction. If the flexion and tap is forcible, this single contraction is followed by others in the ordinary clonus.

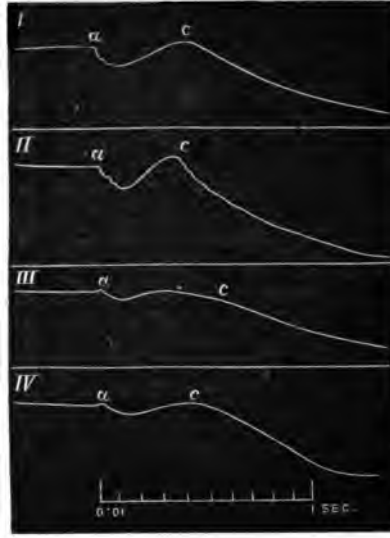
In the case of the tap on the muscle (fig. 7) the interval was in the one patient $\cdot 025$ of a second; in the other case it was $\cdot 045$ of a second. In the case of the tap on the Achilles tendon (fig. 8) the intervals in the one patient with con-

FIG. 7.



Contraction (c) in gastrocnemius excited by tap (a) on the muscle. I and II, case of slight lateral sclerosis; III and IV, case of advanced ditto.

FIG. 8.



Contraction (c) in calf muscles excited by a tap (a) upon the Achilles tendon during passive dorsal flexion of the foot. I and II, case of considerable lateral sclerosis; III and IV, case of slight ditto.

siderable cord disease varied from $\cdot 035$ to $\cdot 04$ of a second; in the other patient, with slighter disease, the interval was $\cdot 04$ second. Thus in the case of the tap on the tendon, just

as conspicuously as in the tap on the front of the leg, the interval before the contraction is insufficient for the occurrence of a reflex process.

But, whatever is the mechanism through which the single contraction is excited by the tap on the Achilles tendon, is certainly the mechanism by which the ankle clonus is excited.

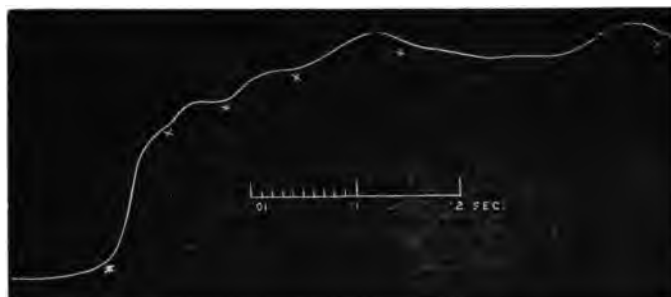
If the theory of a spinal reflex is thus untenable, the theory of stimulation of the tendon, of course, at once falls to the ground. It is hardly necessary, therefore, to point out, that by the tap on the front of the leg the clonus is excited by a mechanism which, however it acts, does not affect the tendon.

The conclusion thus seems to me a necessary one, that the clonic contractions are the result, not of reflex but of direct stimulation of the muscle by the sudden tension or vibration. But this does not exclude all reflex influence in the production of the phenomena. We have to explain not merely the origin of the clonic contractions, but also the origin of the extreme muscular irritability which renders the muscle abnormally sensitive to local stimulation. This irritability is developed by passive flexion of the ankle joint, and ceases as soon as the flexion ceases. That is to say, it coincides with tension of the muscular fibres. The tension may be conceived as affecting their irritability directly, but I think that the phenomena accord best with the hypothesis that the effect of the tension in increasing the muscular irritability is reflex. That such tension is capable of developing a strong afferent impression from the muscle to the nerve centres is shown by an experience which probably has been that of most of us. After a severe attack of cramp in the calf, the muscle is left in a condition of painful sensitiveness. As long as the ankle joint is not flexed beyond a right angle, little discomfort is occasioned by its movements; even a pretty strong contraction of the calf muscles, pointing the toes downwards, occasions little uneasiness. But if the ankle joint is flexed passively, even a little beyond a right angle, the tension on the fibres of the gastrocnemius

occasions acute pain. Moreover, the attempt to obtain the ankle clonus by sudden passive flexion of the foot, occasions a distinctly painful sensation in the calf muscles. It is probable that the centripetal stimulus, which under these circumstances is painful, always occurs during the tension of the fibres, although we are not conscious of it under normal circumstances, unless it is long continued; and that when the reflex irritability of the spinal cord is increased, as in lateral sclerosis, the afferent impression develops in the muscles an extreme degree of readiness to contract on the slightest local disturbance of equilibrium, whether by a direct tap or a sudden increase in tension.

This explanation harmonises entirely with the phenomena to be observed when the development of the ankle clonus, on sudden dorsal flexion of the foot, is followed in its details by taking a tracing such as I now show (fig. 9.) In this, the

FIG. 9.



Gradual development of ankle clonus (x x x) on sudden dorsal flexion of ankle (*). The contractions of the clonus are indicated by the downward movement of the pen. Rapid rate of movement of recording drum. (Reduced to $\frac{1}{4}$.)

first horizontal line is traced by the foot before the passive dorsal flexion was commenced. The latter is indicated by the sudden rise. The first contraction of the gastrocnemius is so slight as merely to flatten and slightly depress the curved line, the second is more marked, and the third more marked still. Thus the irritability of the tense fibres is not instantly developed in full degree by the tension, but is

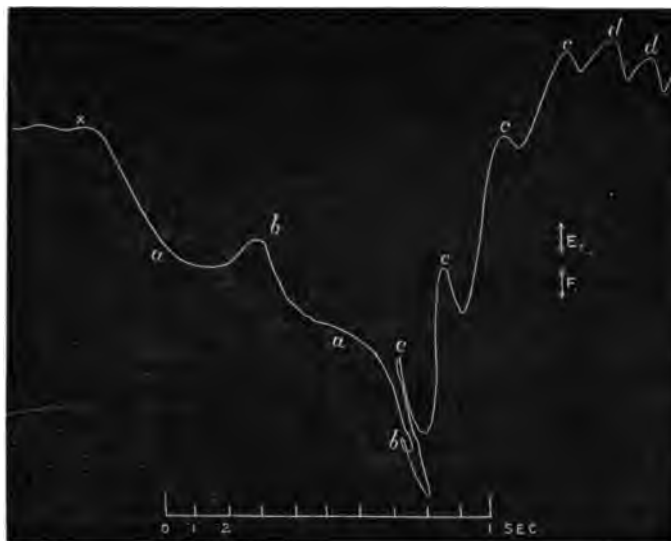
developed gradually, not reaching its full degree until nearly a third of a second after the application of the tension. If the whole were of local production—the irritability as well as the intermitting stimulation—the first clonic contraction should be as great, or greater, in degree, than any of the others, since no stimulation will be as great as that which occurs on the sudden change from complete relaxation to tension. So, too, if the whole of the phenomena were reflex, the first contraction should be as great as any of the others, although delayed until such a time as is required for reflex action. The gradual development of the clonus—which may also be readily seen on simple observation—can only be understood on the explanation now given of a reflex irritability and a local stimulation. If the tension have been previously placed on the muscle, and the reflex irritability thus previously developed, the first contraction from local stimulation is as great as those which succeed, even greater in the case of tap upon the muscle, as in fig. 3.

In several cases I have observed the sudden flexion of the foot to cause, not merely the ankle clonus, but also a reflex contraction of the flexors of the hip, which distinctly *succeeds* the first contraction in the calf muscles.

In the cases in which the ankle reflex is present, there is often also an excess of reflex action in the limbs, which is excited by a painful sensory impression. The form of this reflex action in relation to the action of the gastrocnemius is very instructive. If, for instance, the skin of the leg be pinched, there results, in many cases, a general reflex spasm of the limb, a tracing of which is shown in fig. 10. Below the knee, this does not fall primarily upon the gastrocnemius, but upon the flexors of the ankle joint in the front of the leg. By the strong spasm in these muscles the ankle joint is strongly flexed (*a a*), the foot being brought up, and commonly inverted from the strong action of the tibialis anticus. But this puts the gastrocnemius in a state of tension, and excites in it clonic contractions just as when the hand is pressed against the sole. These occur even while the contraction of the flexors is proceeding (*b b*), but are more

marked when the spasm is subsiding (*c c*), and when it is over they also cease, unless the hand be applied to the sole of the foot during the flexor spasm, when the clonic contractions of the gastrocnemius persist in the ordinary form after the flexor spasm is over, as in the figure (*d d*). This con-

FIG. 10.



Effect of cutaneous stimulation, as pinch (*x*), in causing reflex spasm of limb, flexor spasm predominating and causing strong flexion of the ankle-joint (*a, a*), interrupted (at *b, b*) by clonic spasm in extensors of ankle, which is more marked (*c, c*) as the flexor spasm subsides and continues (passive dorsal flexion being maintained) as the ankle clonus (*d, d*) when the flexor spasm is over. Before the peripheral stimulation the hand was gently pressed against the sole, and slight ankle clonus occurred, indicated by the waves in the initial line, although not visible to the eye.

stitutes a paroxysm of the one form of so-called spinal epilepsy, of the most typical form to which the term can be applied. The clonic spasm in the gastrocnemius, excited by, and super-added to, the general spasm, predominating in the flexors, constitutes the chief part of the clonic phenomena of the paroxysm, and being most distinct during the subsidence of

the tonic (flexor) spasm, gives to the phenomenon its resemblance to an epileptic convulsion, which was pointed out by Brown-Séquard, a resemblance which is thus wholly superficial.

It is always this form of spasm which is excited by *pinching* the tendo Achillis; but I may remark that careful observation may be necessary to distinguish this fact, since the clonic spasm in the gastrocnemius begins, as is seen in the tracing, during the rise of the flexor spasm, and it may begin so soon after the commencement of the latter, that it may easily be mistaken for a primary effect. The flexion of the foot is always, as in the spasm shown in the tracing, the initial event.

What, then, is the explanation of the knee clonus (p. 289) which is set up, during passive extension of the patellar tendon and extensor muscle, by a tap on the tendon? The contractions, as fig. 5 shows, succeed one another at a much longer interval than the contractions of the ankle clonus. This is in proportion to the longer interval which elapses between the stimulus and the contraction, in the case of the ordinary patellar reflex, than in the single contraction of the extensor of the ankle. The proportion between the frequency of the ankle clonus and the frequency of the knee clonus is nearly as 4 to 10: and the proportion between the interval which intervenes between a tap on the Achilles tendon, and the contraction of the gastrocnemius, is to the interval between the tap on the patellar tendon and the contraction of the thigh muscles also as 4 to 10. It has been seen that the difference of interval in the case of the isolated contractions affords ground for the conclusion that the mechanism of the two is different. The same conclusion is suggested by the corresponding difference in time in the two forms of clonus, viz. that the several contractions of the knee clonus are of true reflex origin. The sudden tension of the quadriceps by the weight of the extended leg on the fibres, after the cessation of one contraction, probably constitutes the afferent stimulus for the next. It will be remembered that the single contraction may be excited by a blow on the tendon above the patella, and even by a blow on the muscle,

as well as by a blow on the tendon below the patella. It seems probable, therefore, that the individual contractions in the quadriceps owe their origin to the mechanism which develops the irritability to local stimulation in the calf muscles.

Should the objection suggest itself, that the difference in time between the knee and ankle clonus is due only to the larger size of the muscles which move the knee, I would point out that the difference in size between the calf muscles and the plantar toe muscles is incomparably greater than that between the calf and the thigh muscles, and yet the ankle clonus and the toe clonus have practically the same time.

In the case of the ankle and toe clonus the conclusion is, that the passive extension leads to a reflex irritability, a hair-trigger susceptibility to local influences, an incipient contraction, excited to developed contraction with extreme readiness, and that this irritability is at its maximum immediately after the cessation of a previous contraction. It is, I think, an interesting and significant fact that, in these two sets of muscles, those which extend the ankle and flex the first phalanx of the great toe, the sequence of passive extension and tonic contraction is being constantly developed under ordinary circumstances in the act of walking. In each step, after the leg is put forward on to the ground, first the ankle joint is flexed by the anterior tibial muscles, and then, when the calf muscles have thus been made tense, they pass into a state of tonic contraction, by which the heel is raised, and simultaneously the great toe muscles (previously extended by the flexion of the toe) also contract, as the foot rests on the ball and great toe and the body is impelled forwards. This sequence of tension (which has been seen to be attended with an afferent impression) and tonic contraction is frequently repeated—with persons who lead active lives not less than fifty thousand times every day—and the development of a reflex mechanism between the afferent and sequential efferent impulses, will be almost a necessary

result. It is this reflex effect which is assumed to be so excessive in lateral sclerosis of the cord as to permit the local development of the ankle clonus. I have never been able to obtain the ankle clonus in a child who had never walked, although such children not rarely exhibit a form of paraplegia similar to that in which, in older persons, the phenomenon occurs. It may be supposed that the act of learning to walk opens up the reflex path between tension and contraction of the calf muscles. We are able thus to understand why the ankle clonus, although of local excitation, occurs in cases in which the reflex action, as shown by the knee reflex, is in excess.

The phenomenon which I have described as the "front-tap contraction" is, I think, of considerable practical value. It is an exceedingly delicate test of the reflex irritability in the calf muscles, and may be obtained when their irritability is insufficient to permit the development of the clonus. I have found it in cases before the clonus could be obtained, and in cases which improved after the clonus had ceased to be obtainable in the ordinary way. When once it can be produced, if there is no ankle clonus, there is always a great excess of the knee reflex. It is occasionally, though not usually, to be obtained in apparently healthy individuals, and so is, alone, of less absolute value than the ankle clonus. In those who present it, however, although the power of the legs may be good, there is always an excessive knee reflex.

It has been remarked that, as a rule, if the ankle clonus can be obtained, the knee reflex is excessive. In such cases the disease is above the lumbar enlargement or in the lateral columns, and leaves both reflex processes unimpaired. But since the knee reflex occurs through nerves which arise higher up the cord than those through which the reflex irritability of the gastrocnemius is developed, it is conceivable that a local lesion in the upper part of the lumbar enlargement might abolish the knee reflex, and yet lead to the development of the ankle clonus. This combination was, in fact, presented by the following instructive case:

CASE 5.—John W—, æt. 44, came under my care at the National

Hospital for the Paralysed and Epileptic, November 11th, 1878. Weakness in the legs, chiefly in the left, commenced nine months before, and in a few weeks had reached such a degree that he could not move the legs at all. Power was slowly recovered, until his state on admission was reached; then he could only just stand with assistance. After a few weeks' treatment he had recovered so much power that he could walk half a mile. His condition then was as follows: Arms normal. When lying he could move the legs freely at all joints, but the left with less power than the right. The left quadriceps extensor was thinner than the right, and its irritability to faradisation was lower; it required two centimeters more of the secondary coil of Stöhrer's larger battery than the right, while it contracted to two cells of the voltaic battery less than the right. There was a similar difference between the two glutei. The flexors of the knee and all the muscles below the knee were equal in nutrition and irritability on the two sides. Sensation in the right leg was normal. In the left leg in front of the thigh and in the part supplied by the ilio-inguinal nerve a touch was felt at once. On the front and outer part of the thigh, in the part supplied by the anterior crural, middle cutaneous, and external cutaneous, neither a touch nor prick could be felt. Sensation was good on the outer part of the leg below the knee, in the part supplied by the external popliteal (sacral plexus), but not in the inner part (supplied by the internal saphenous of the lumbar plexus), until near the ankle. On the foot, back of leg, and back of thigh, the parts supplied by the sciatic, it was normal. There was also a little diminution of sensation over the whole of the left gluteal region supplied by the small sciatic. Cutaneous reflex from the sole of the foot was very marked. In the left leg typical ankle clonus could be obtained; in the right foot no clonus, but a single contraction, with the foot in passive dorsal flexion, on striking either the Achilles tendon or the front of the leg. Patellar tendon-reflex well marked on the right side; absent, absolutely, on the left.

There was thus diminished sensibility in the parts supplied by the external cutaneous (2nd lumbar nerve) and middle cutaneous and anterior crural (2nd, 3rd, and 4th lumbar), and gluteal region (supplied above by the 2nd lumbar, and below by branches from the small sciatic, which is almost certainly derived by the lumbar sacral cord from the 4th lumbar nerves). There was diminished irritability and some wasting in the quadriceps extensor femoris, supplied by the

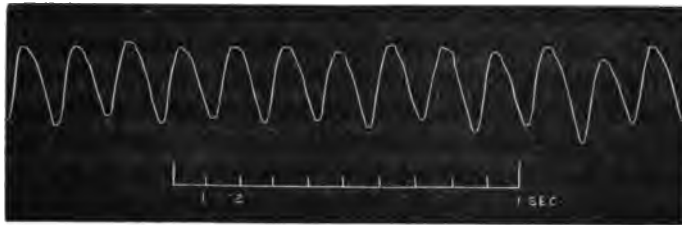
anterior crural (2nd, 3rd, and 4th lumbar) and the gluteus maximus (small sciatic, probably from 4th lumbar), while there was no impairment of sensibility or wasting in any part supplied by the great sciatic, into which the 5th lumbar and sacral nerves go. There is thus strong reason to believe that there was a lesion, perhaps due to pressure from syphilitic disease, of the left side of the cord, and to a much slighter extent of the right side in this region. It had arrested the knee reflex, but the unaffected portion beneath had undergone the same increase in reflex activity which the whole lumbar enlargement presents in disease of the dorsal region.

In the cases in which the ankle clonus can be obtained, there is often a considerable amount of general muscular spasm in the legs, excited by sensory impressions or by attempts at voluntary movement. When this general spasm is considerable, the condition is that which has been designated "spastic paraplegia," "spasmodic tabes dorsalis," &c. When the spasmodic action becomes great, the ankle clonus often can no longer be obtained. The attempt to elicit it produces general tonic spasm in the limbs. I have, in several cases, observed the gradual cessation of the ankle clonus as the spastic state became developed. In some cases it could still be obtained at periods when the spasm was slight. It is probable that the reflex tonic irritability in the gastrocnemius, which permits the ankle clonus, is part of the same reflex action which in greater degree, or so to speak, storm-like action, leads to the tonic spasm in which the local stimulation cannot produce its effect.

The question suggests itself—if the reflex mechanism by which the ankle clonus can be developed by local stimulation is a normal phenomenon, excessive in lateral sclerosis, would not the ankle clonus also be obtainable, in slighter degree, under normal conditions? I have found that the ankle clonus is to be obtained in many, probably in most, individuals under normal circumstances. It cannot, indeed, be produced by passive dorsal flexion of the foot, but it may be obtained in another method. The ankle clonus in

spinal sclerosis may be produced very readily by making the patient sit on the edge of a seat with the knee flexed at a little more than a right angle, so that the foot rests on the ball, and the heel does not quite touch the ground. This posture evidently involves tension of the gastrocnemius. If then the top of the knee be tapped, the tension on the gastrocnemius is suddenly increased, and the ankle clonus is set up. The occurrence of the movement on this posture has been remarked by Dr. Buzzard, and was indeed observed long before the ankle clonus excited systematic attention. If a normal individual assumes this posture, a tap on the knee does not set up the movement; but if the movement is initiated by a voluntary effort, it will go on, often increasing in degree, without the slightest voluntary action, as long as the posture is maintained. A tracing from such a movement (fig. 11), obtained by attaching the writing lever to the

FIG. 11.



Normal ankle clonus from a healthy individual.

knee, has the same characters as that in spinal sclerosis. It is as regular, and occurs in just the same time. I show four tracings obtained from the first four individuals whom I examined—two strong robust hospital porters, the resident medical officer at the hospital, and myself. The time varied a little in the several instances, but within the limits of variation presented by the ankle clonus. In one case the number of contractions per second was 7, in the second 6, in each of the others 6.2 per second. How uniform the movement is, is shown by the fact that in one individual two observations gave respectively in equal periods of 9 seconds, 63 and 64 contractions, and in another, two ob-

servations on the one leg gave 56 and 57 in the same period and in the other leg 56 contractions.

These facts suggest that a reflex relation between muscular tension and contraction may play an important part in the mechanism by which often-repeated movements are co-ordinated. This reflex is excessive in lateral sclerosis of the cord. It is probably lost in posterior sclerosis, locomotor ataxy. The allied knee reflex is lost, and I have found that the normal ankle clonus cannot be produced, nor any voluntary movement resembling it.

The phenomena of muscular inco-ordination in this disease may be in part due (as Tschirjew also has suggested) to the loss of this reflex between tension and tonic contraction. In such respect the condition of the ataxic will resemble that of the infant who has never walked, and it has often been remarked how similar is the unsteadiness of the one to that of the other.

Within the last few days Mr. Romanes has communicated to the Royal Society observations which prove that, in the *Medusæ*, rhythmical movements result from a *continuous* nervous stimulation, the alternate exhaustion and return of muscular irritability determining the rhythmical character of the movement. The theory of the ankle clonus, which the observations, above recorded, suggest, is also that of a continuous nervous stimulation, but the rhythmical movement occurs as a consequence of the continuous central stimulation, and the continuous local stimulation of passive tension. The muscular contraction excited is transient, and the local stimulus only affecting the fibres when they are not contracting, the contraction, as it were, interrupts the stimulus, and renders the continuous influence an intermitting stimulation. The phenomenon thus indicates another method by which rhythmical action may be the effect of a continuous influence.

ON
OPHTHALMOPLÉGIA EXTERNA
OR
SYMMETRICAL IMMOBILITY (PARTIAL) OF
THE EYES, WITH PTOSIS.

BY
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THE cases to which I have to ask the attention of the Society are characterised by a very peculiar group of symptoms. Drooping of the eyelids, so as to give to the face a half-asleep expression, is usually the first, and it is soon accompanied by weakness of all the muscles attached to the eyeball, so that the movements of the latter become much restricted, or even wholly lost. The condition is usually bilateral, though it is not always exactly the same in degree on the two sides. Its symmetry probably denotes that it is of central origin. It by no means always happens that all the ocular muscles are alike affected, or that they are attacked simultaneously, still it is a very marked feature of the malady, that the muscles fail in groups, and not singly. Non-symmetrical paralysis of single ocular muscles is, of

course, very common, especially in connection with syphilis and locomotor ataxy, but such cases are to be distinguished from those which I am now describing: first, by the fact of non-symmetry: secondly, by the early completeness of the paralysis; and, thirdly, by the ease with which, very frequently, they are cured. In a majority of them, there is, perhaps, good reason to suspect that a gumma in the nerve trunk is the cause. In the symmetrical cases now under consideration, however, the changes probably begin centrally; they are usually slow in progress, and are often difficult of relief. They agree with the single nerve cases in that they occur chiefly to those who have had syphilis. Although I have ventured to speak of immobility of the eyeballs, I by no means wish to imply that it is usually complete. On the contrary, incompleteness in the degree of paralysis is almost as marked a feature as is the tendency to affect many muscles at the same time. Although the eyelids droop, there is seldom complete ptosis; great limitation of the range of motion of the eyeballs is more common than fixation. The degree, however, varies with the stage, and at a late period the paralysis may be absolute. The third, fourth and sixth nerves are of course those which are involved, but not unfrequently in the early stage one or more of these may wholly escape. Occasionally the optic nerve itself is involved, and sight is lost. I am making these statements from the observation of only a limited number of cases, for the condition is but seldom seen. I have as yet witnessed only a single fatal case, and in it evidence was afforded of extension of disease downward into the motor tracts of the cord, and the man lost the use, first of his upper, and subsequently of his lower extremities.

In a former paper¹ I have ventured to propose the name *Ophthalmoplegia Interna*, for cases supposed to depend upon disease in the lenticular ganglion, in which the internal muscles of the eye (the iris and ciliary muscles) are together involved in paralysis. If in contradistinction with these, I may now be permitted to suggest that of *Ophthalmoplegia*

¹ 'Med.-Chir. Trans.,' vol. lxi, p. 215;

Externa for those in which the external muscles of the eyeball fail in power, it must be with the full admission that these latter often, indeed usually, include the internal muscles also. With paralysis of the third nerve, the circular fibres of the iris and the ciliary muscle must of course fail, but the defect in these parts in this class of cases is, usually, as I have asserted of the recti and levators, incomplete. In this incompleteness, at any rate in the early stages, it differs from what we see in cases of supposed disorganisation of the lenticular ganglion. Nor does it always happen, even when in ophthalmoplegia externa the third nerve root to the ciliary ganglion is involved, that the vaso motor filaments suffer too, and in this we find a further difference from what would be expected if the ganglion itself were attacked.

The cases in question are probably closely allied in nature to what is known as progressive muscular atrophy, their peculiar feature being, that only one special set of muscles (or rather nerves) is at first attacked. We have, probably, in them, a very close parallel to the so-called bulbar paralysis, the labio-glosso-laryngeal paralysis of Duchenne. In it, as in ophthalmoplegia externa, central degeneration-changes occur, and the result is the paralysis of a set of associated muscles. It may be plausibly conjectured, that the initial lesion is inflammation of the nuclei of the affected nerves, which, in a slowly serpiginous manner, creeps from place to place, along certain definite anatomical paths. Within certain limits its directions of spreading, and its progressive tendency may vary in different cases, but speaking generally, the cases are remarkably same in their features. In exceptional instances, definite symptoms of locomotor ataxy are present, and in others still more rare, the fifth nerves, or the seventh, or even the eighth may be involved. Although, so far as I am aware, this group of cases has not as yet found any record in English medical literature, it has not escaped notice in Germany. Graefe described examples of it in 1867, and employed the word ophthalmoplegia in reference to them, and Eulenberg in his work on 'Functional Nerve Disorders' devotes a short chapter to the subject, which, however, con-

sists chiefly of an abstract of Graefe's communication. By both these authors it is spoken of as sometimes syphilitic, and sometimes rheumatic, and occasionally without assignable cause.

The clinical part of my paper consists of the narrative of fifteen examples of the malady. Several of these cases extend over many years, and are very detailed. I have done my best at abbreviation, but am very conscious that I shall still need the Society's indulgence in this matter.

CASE 1.—My first case occurred in 1862, and has special interest, because I am able to produce a photograph of the patient. A printing-house porter, aged 52, applied at Moorfields almost blind with white atrophy of the optic nerves. His eyelids drooped a little on both sides, and he was obliged to elevate his head, and use the occipito-frontales in order to see. The eyes were almost fixed in the orbits. The photographer to whom I sent him, reported him "an unusually good sitter, for he never moved his eyes." Both eyes diverged, the left especially. On both sides all the third nerve muscles were paralysed completely, excepting the levator. On the left side, the fourth nerve was paralysed, and the only muscle which he retained was the external rectus. On the right side, the sixth nerve was paralysed, and the only muscle which he retained was the superior oblique. In both eyes the discs were white and the retinal arteries small. He was not deaf, his faculties, excepting sight and smell, were good, and he had excellent health. He had never had "a fit" nor any limb paralysis, and he had never suffered from pains in his head.

The case was remarkable on account of the long interval between the paralysis of the muscles, and the failure of sight. The progress of symptoms in the early stages appeared to have been very slow. The history as to syphilis was that in 1834 (that is thirteen years before his first eye-symptoms) he had a long treatment by mercury for a chancre with buboes. He was salivated. No secondary symptoms were remembered. In addition to the eye-symptoms his smell had failed,

Ammonia made him sneeze, but he could detect no odour in hyoscyamus. He considered his taste perfect.

This man remained under my observation from January, 1862, to July, 1864. In August, 1863, he began to complain that fluids in swallowing came through his nose. His speech was thick, and the palate appeared very deficient in sensation. His pupils were quite motionless, and of medium size. His uvula could not be made to move. In July of 1864 he was in the same state. The left external rectus, and the right superior oblique, still acted well. The muscles of his palate did not act, but tickling it, although but little felt, caused sickness. His cheeks hung loose as if partially paralysed, but nothing positive could be proved. He could still whistle.

After 1864 this patient ceased to present himself. From inquiries at the workhouse I believe that he died in 1866.

CASE 2.—My second case is that of a young physician who, in 1871, put himself under my care for symmetrical paralysis of the third nerves. He was thirty years of age, and had been treated seven years before for a chancre (by the late Mr. Gascoyen). No secondary symptoms followed, but he had since had frequent reminders in the form of psoriasis palmaris, &c. He had also been liable to "rheumatic pains," which were always relieved by iodide of potassium. About a week before I saw him he began to be troubled with morning sickness, then his eyelids felt heavy, and next they drooped, and he began to see double. When he came to me he had slight ptosis on right and complete on the left. On the right the pupil was dilated and fixed, and accommodation lost; whilst in the left the pupil was normal and accommodation was perfect. The internal rectus was very defective on both sides. On the left the superior and inferior recti were almost wholly paralysed, whilst the superior oblique was perfect. On the right these recti were weak, but only to a slight degree, but a fortnight later the ptosis on this side also became complete, and all the recti, excepting the external, were absolutely paralysed. Both eyes diverged, and, on the right side, he had entire loss of accommodation,

At this period he complained much of "horrible muscular pains" in his legs, which were relieved by exercise. He had also morning sickness, almost exactly like that of pregnancy, and some frontal headache. Subsequently there was general dulness of sensation in the lower extremities. Dr. B. remained under drug treatment almost continuously for two years and a half. We gave mercury in various forms, and the iodide in very diverse doses. Sometimes, after an interval, five-grain doses produced very definite results, and at one period he had got up to *an ounce and a half in the twenty-four hours* without feeling any discomfort beyond lassitude. The sum of our experience was, that the iodide did more good than mercury, but that it was useless unless the dose was frequently and liberally increased. The functions of accommodation and the use of the internal recti varied much from time to time, and always afforded us a good test of the efficiency of treatment. I never, in any other case, pushed the iodide to anything like the extent to which it was given in this instance. The result, however, was most satisfactory. Dr. B. lost all his ataxic symptoms, he regained perfect accommodation, and most of the ocular muscles recovered their power. He is now, seven years after the conclusion of treatment, in good health, and has never been threatened with any relapse. It should be added that we had the advantage of sea-air at the time that the largest doses were given, and without its aid I much doubt whether the result would have been as satisfactory as it was.

CASE 8.—The following case was under my observation from its commencement, in 1869, to its end, in 1876, and it is of especial value as the only one in which I have obtained a *post-mortem*. The symptoms, which began with ocular paralysis and amaurosis, were slowly progressive, and the case in part resembled locomotor ataxy, and, in part, progressive muscular atrophy.

Robert S—, æt. 48, a very healthy-looking man from the country, formerly a policeman, now a gardener, was sent to me by my late colleague, Mr. Dixon, in March, 1869. He

had a slight defect of his right sixth nerve, and could not abduct the globe well. There was slight convergence, and he complained of seeing double. The right pupil was rather larger than the other. He complained that he did not see well by artificial light, and that he could not read long at a time. He had enjoyed good sight until six weeks before, and had never used glasses. I found that without glasses he could, by effort, with left eye read No. 10, but with right only 14. For a minute or two he could puzzle out smaller print, and it appeared that his defect was wholly due to weak muscles of accommodation, for with +16 glass he read No. 1 easily with either eye, and his distant vision was almost perfect.

I could not arrive at any opinion as to the cause of the rather sudden onset of symptoms. He denied having ever had syphilis, and he had been married early and had seven living children. He had never had any injury to his head more serious than the blows to which policemen are liable, nor had he had gout. Of late he had suffered a good deal from what he called "rheumatism" between his shoulders and had also had giddiness and much pain across his forehead. I prescribed iodide of potassium, and during a six weeks' treatment with it he made some improvement, and I then lost sight of him. I am sorry that I cannot state the exact degree or kind of improvement, but he became able to continue his work as gardener with comfort, and, as he lived at a distance, he was anxious to avoid journeys to town.

Four years later, in October, 1873, Robert S— again came to me at Moorfields. In the interval his sight had steadily deteriorated, and with the right eye he had now only perception of light, whilst with the left he could see small spell No. 20. He could not abduct either eye, and the right was habitually crossed inwards. There was no paralysis of any of the other ocular muscles, but they all acted feebly, and he had the same sleepy look observed in other cases, from not being able to keep his upper eyelids properly elevated. He could, however, by effort, lift his eyelids. His left internal rectus seemed weaker than the right. The optic

discs were both very pale, and the artery and vein much reduced in size. The atrophy was more advanced in the right. He still enjoyed good health, and, in spite of being almost blind, still acted as gardener and groom. I could not make out much as regards other evidences of disease of the nervous system. He was liable to attacks of severe pain in his forehead, sometimes lasting a week or more. For seven or eight years he had been liable, he said, to cramp in his legs at night. His bowels were constipated, but not extremely so. At this date his pupils did not act in the least—the left being of medium size, and the right rather larger. He had no habitual headache. I now prescribed mercury and pushed it to ptyalism, but with no definite benefit.

During the spring of 1874 I saw him repeatedly in the country, and about March the following note was made:—Pupils motionless. External recti paralysed, and all the others imperfect. The superior oblique appear to be also weak, but as he can still use the inferior recti a good deal it is difficult to test them. His bowels have been very costive, and he complains much of tightness round the abdomen as if a strap were round him, and also of numbness in the skin of abdomen and face. There is also slight numbness in hands and a little in feet. Up to this date the man had been able to get about, and used to drive himself in a pony carriage a distance of seven or eight miles to see me. Not long afterwards, however, an aggravation of symptoms took place and he became confined to his house. After this I did not see him again. I was informed that he had a “sort of choking fit” and soon after became absolutely blind. His extremities next failed him, and he had to keep his bed. He now suffered from dreadful pain in his head, and was frequently “out of his mind,” and liable to use most violent language. He could speak well and swallow easily, and used to eat largely. His lower extremities became quite useless and were usually “icy cold.” In this condition he died in May, 1876, about seven years after the commencement of his symptoms.*

* The following particulars were kindly obtained for me by Dr. Sloman, of Farnham. “About Christmas, 1874, loss of power in his arms was

Two very remarkable coincidences occurred in this case and by their aid I am enabled to complete its narrative. They are so peculiar that I must ask the Society's excuse if I briefly mention them. I had been very desirous to know whether this man had suffered from syphilis; as stated, he stoutly denied it, and he appeared to be straightforward. I got him to bring me two of his children and neither of them showed anything in the least suspicious. So matters stood when in April, 1876, Dr. Horace Jeaffreson of Wandsworth wrote to me that he had under his care a maid-servant who was the daughter of a man in whom I had taken much interest and that the girl was now suffering from inflamed eyes. He sent to me Robert S—'s eldest child, a girl of twenty, with *notched teeth and a most characteristic condition of syphilitic keratitis*.

This girl, who became my patient at Moorfields, informed me from time to time of her father's condition. I was very anxious to get Dr. Hughlings Jackson to see him, and for a year or more we had it in contemplation to go some day to Farnham and ascertain the present state of his symptoms. One Saturday afternoon in the spring of 1876 we accomplished our intention, and having found the man's cottage ascertained that he had died the day before. After much persuasion we succeeded in getting permission for a post mortem, and brought his brain home with us.

Dr. Gowers was kind enough to make for me a detailed microscopic examination of the cerebral nerves and their nuclei, and I append his report. It will be seen that degenerative changes precisely similar to those of progressive

noticed; he would drop a cup or anything he was trying to drink from. In January, 1875, his legs became weak, and he took to bed at the end of January or beginning of February; he was constantly in bed after that. During this time his arms and legs would from time to time "fly up" (as his wife expresses it), and as suddenly drop again. His lower limbs were constantly cold. He had no constant paralysis of the sphincters, but very occasionally urine was passed unconsciously. The bowels never acted at all without medicine. He could swallow perfectly, and also speak and taste. He had no paralysis or loss of sensation in muscles of face. He used to suffer much from headache."

muscular atrophy were found. These changes implicated the optic nerves and the third, fourth, and sixth. The fifth were slightly affected, but all the others were healthy. We had not been able to obtain any part of the spinal cord. From the nuclei of all the nerves mentioned the cells had disappeared.

Examination of the brain by W. R. Gowers, M.D.

The brain, when received, was somewhat softened from commencing decomposition, and the following facts are all that could be ascertained with certainty. The cranial nerves were examined microscopically, in the fresh state; the pons and medulla after hardening.

Nothing abnormal was observed in the convolutions. The corpora striata were apparently normal, the only exception being the presence of a hyperæmic patch in the left lenticular nucleus. The optic thalami were of normal size and consistence, except that the posterior tubercles were perhaps a little smaller than normal, and a little softer. Microscopical examination did not show any special change. The corpora quadrigemina were of nearly normal size; the posterior only being distinctly smaller than natural, but on microscopical examination they appeared healthy. The pons and medulla were of the usual size, and the only abnormality was an unusually deep central sulcus of the floor of the fourth ventricle, but this was found, on further examination, to be independent of any change in the structure of the pons.

Cranial nerves and their nuclei.—The *olfactory nerve* appeared normal. The *optic nerves*, and chiasma were uniformly grey, but of fair consistence. The optic bracts were also grey, but in places presented a white almost nacreous striation. Microscopical examination of the nerves and tracts showed many fat globules and degenerating fibres, but also a large number of healthy fibres. *Third nerves.*—Smaller than natural, grey and translucent. Very few healthy fibres could be seen in them; some fibres were seen undergoing degeneration, and there was a large number of

connective tissue nuclei. In the crura cerebri the passage of the tracts of fibres of origin was indicated by lines of connective tissue fibres in which scarcely any nerve fibres could be seen. Their nuclei beneath the nates presented striking changes. Almost all the large multipolar nerve cells had disappeared, two or three only were to be seen in each section. A few cells were seen of some size, but without processes. Others appeared represented by minute angular cells not larger than connective tissue nuclei. The latter were very abundant throughout the tract.

Fourth nerves.—No trace of these could be seen. They had probably become reduced to fine connective tissue threads, indistinguishable from the fibres of the pia mater. Their nuclei beneath the testis presented a similar degeneration to those of the third nerves in the anterior part of the same tract.

Fifth nerves.—The upper fibres of the large root appeared healthy, but the lower fibres had a grey appearance and presented granular degeneration and segmentation on microscopical examination. Within the pons there was little recognisable alteration in the fibres of the nerve, and the nucleus was for the most part normal. Here and there were patches of disintegration. The nucleus of the motor root of the fifth was in all respects normal.

Sixth nerves.—These were reduced to fine grey threads in which, under the microscope, scarcely a single nerve-fibre could be seen. They were made up of nucleated connective tissue, with here and there a row of granules to indicate the position which had been occupied by a nerve fibre. Within the pons lines of connective tissue alone indicated the course of their tracts of origin to the nuclei beneath the eminentiæ. These, the so-called "conjoined nuclei" presented general degeneration; most of the large nerve cells had disappeared and only granules, nuclei, and small angular cells remained.

Facial nerves.—Perfectly normal, both in their trunks and roots of origin within the pons.

Auditory nerves and nuclei normal.

Glosso-pharyngeal nerves and nuclei normal.

Pneumogastric nucleus normal.

Hypoglossal nucleus normal.

Throughout the medulla, pons, and corpora quadrigemina the perivascular erosions, so frequently met with, were very large and numerous. In the lower part of the floor of the fourth ventricle there were some areas of disintegration, in the grey substance just beneath the lining membrane; and the surface, partly from this cause, was more than usually irregular. One such area appeared to have been caused by a small hæmorrhage.

Remarks.—The degeneration of the nerve roots, and disappearance of the nerve cells from their nuclei of origin, are precisely similar to the changes seen in other parts in progressive muscular atrophy. There was no indication of pressure upon the nerves, nor of any acute change in their nuclei, nor in any centre. The disintegration and connective tissue changes are constantly met with in the grey matter of the cord in progressive muscular atrophy.

CASE 4.—In the following case no history of syphilis was acknowledged. It was, however, very closely similar to the others which were syphilitic; and when we remember what occurred in the preceding case in this matter, we cannot trust much to negative statements.

Abstract.—Almost complete paralysis (not equal) of all the ocular muscles, with the exception of the inferior rectus on right, and external and inferior on the left. Slow failure of sight in both eyes, but not symmetrical. Atrophy of discs with but little diminution of central vessels. A feeling of tightness across forehead and great irritability of temper. Patient a man of middle age in good health. Insanity threatened.

William M—, a florid healthy-looking man, æt. 45, came under my care in January, 1875. He considered that he ailed nothing whatever excepting his loss of sight. His pupils were of medium size and quite motionless. With his left eye he could see a hand, but could not count fingers, whilst with the right he could just spell out No. 20. His

manner was slow and dull; he answered me in monosyllables, and his wife said that at home he would often sit for hours and not speak. He slept much. He complained occasionally of dizziness, and said that he frequently had "a sort of tight pain" across his forehead. He had no pain in his limbs or joints. The account which he gave of his first failure of sight was, that it occurred when he felt quite well. He began to find his eyes dull, and they felt as if sand were in them. He had no sickness and could walk well. The left eye failed first. He attended at Guy's Hospital in 1873, and took phosphorus and nuxvomica. A note on his Guy's paper, stated that "the optic discs were white and atrophic, blood supply to retina good." In June, 1874, he had an eruption on his legs, which he thought was due to the medicine. He describes it as purpuric.

When he came to me his expression was somewhat sleepy, from his not opening his eyes well, but there was no positive ptosis. The left globe oscillated somewhat from side to side. Many of his ocular muscles were paralysed. Thus, on the right side, the only muscle which enjoyed any material power was the inferior rectus. The others, although not absolutely paralysed, were almost so. On the left side the external rectus acted well, and the inferior fairly, but all the others only very slightly. When told to look upwards he simply converged his eyes. None of the muscles were absolutely paralysed, excepting, perhaps, the right external rectus.

The pupils dilated well with atropine. The optic discs were both of them pale, the left being much whiter than the other. The branches of the artery were but little diminished.

M. was by occupation a dock labourer, but he said that he often did not work more than two days a week, as he was lame in consequence of a compound fracture of one leg. He had been well fed and had a good appetite. He had always been temperate in stimulants, and had never smoked. His smell, taste, and hearing were perfect. He denied having ever had syphilis, or that he had experienced any sexual

failure, but in these matters he was not communicative. He had married, for the first time, eight years before I saw him. His wife was older than himself.

In March of same year he was in much the same state. His club doctor had certified that he was insane; but his wife denied it, and although irritable, he had always to me appeared rational.

CASE 5.—The case of F. W. M—¹ is a very characteristic one. He was an engine-fitter, æt. 39, in apparently good health, and free from symptoms of spinal disease. He had suffered from complete syphilis twenty-two years before the eye symptoms began. The latter, although ultimately symmetrical, were not so at first. In the beginning all the recti on the left side failed, the pupil became dilated and fixed, and accommodation was lost. Amaurosis on the same side followed, and two years later similar symptoms were developed on the right side, and in the same order. Under a prolonged but perhaps too mild anti-syphilitic treatment his disease advanced very slowly. Six years after his admission, and nine from the commencement of his symptoms, he was quite blind, and his eyes almost fixed. He still possessed, however, slight power over certain of the recti. Excepting some occasional shooting pains in the head, and a degree of numbness of the forehead, he had not experienced any other nerve-symptoms, and was when last seen in good general health.

CASE 6.—The case of John H—² a labourer in wine-vaults, æt. 34 is of special interest, because symptoms of locomotor ataxy preceded those of ophthalmoplegia. It was not certain that he had ever had syphilis, and excess in venery, alcohol, and tobacco were amongst the possible causes. Obstinate constipation, retention of urine, impotence, and weakness of the lower extremities, were his earliest symptoms. Then followed symmetrical amaurosis and paralysis of the recti muscles on both sides. The pupils

¹ Detailed notes of this case extending over nearly six years were appended to the paper.

² Notes of this case also accompanied the paper.

had become dilated and motionless before the ocular muscles failed. He ultimately became almost blind.

CASE 7.—The next case which I shall mention is the last which has come under my notice, a very definite example, and one in which I had the pleasure of producing the patient for the examination of the Society on a former occasion. The patient is a butler in a country house. He is thirty-four years of age, and about ten years ago he went through a sharp attack of syphilis. He has now been for three years or more the subject of ophthalmoplegia externa in combination with some symptoms of ataxia. I have had him under treatment for about a year, and under specifics in full doses he has greatly improved, but the movements of his eyeballs are still in all directions much restricted. None of the muscles are absolutely paralysed, but all are very feeble. It is important to note that his pupils act fairly, and that his accommodation is perfect. His lower extremities were, at one time, very weak, and liable to much aching pain. He walked badly, and used occasionally to fall. These symptoms have now passed off, and he walks well, and appears to be in excellent health. This patient had taken the iodide before I saw him, and there is every reason to believe that it has been the means of saving his life, for, at one time, the symptoms were rapidly progressive, and he was confined to bed.

CASE 8.—Mr. Ilott, of the Bow Workhouse Infirmary, sent me in 1878 an interesting example of this malady in connection with a syphilitic history, and with modified symptoms of locomotor ataxy. The patient was an actor, æt. 30, of much intelligence, who had already been under hospital treatment, and who told me spontaneously that he had suffered from syphilitic disease of the brain. He had had syphilis eight years before, and at the time that his nerve symptoms began he was under Mr. World's treatment for a tertiary ulcer on the leg. The onset of his symptoms was, according to his account, sudden. He one day felt a "click" in his head and fell down in the street. He got up,

walked to an omnibus, and went home, but ever afterwards one eyelid drooped; a few weeks later the other eyelid drooped, and for a time both eyes were closed. Under treatment by iodide of potassium, extending over a year, he recovered, and when he came to me he considered himself well, and was not taking medicine. I found his pupils motionless, and of medium size, but accommodation almost perfect in both. His lids drooped to a moderate degree, and, excepting the internal rectus on each side, almost all his ocular muscles were more or less paralysed. The conditions were almost symmetrical. He could see well, and suffered no pain. His gait was decidedly ataxic, and he complained that his legs were weak. The sexual function was almost wholly in abeyance.

The subject of the above case was presented before this Society last year, and a week afterwards Dr. Gowers was kind enough to send to me for examination the subject of the following as an example of similar disease.

CASE 9.—A servant girl named B—, æt. 27, had been under Dr. Gowers' treatment for several years. Four or five years ago, after much headache, both eyelids began to droop, and subsequently the ocular muscles on both sides became involved. Her pupils continued to act well, and she retained sight and accommodation. At the time of my examination almost all the muscles were defective, and some almost completely paralysed. The inferior recti and the inner rectus on right side had most nearly escaped. There were some indications of ataxy. Her lower limbs were weak, and she walked badly, complaining that she could not go up stairs easily, and that sometimes her legs seemed to give way. Once she had tripped and fallen. I did not ask direct questions as to syphilis, and there was no other clue to it.

I am indebted to my colleague, Mr. Waren Tay, for the notes of two cases in which young children were the subjects of symmetrical paralysis of the muscles of the eyeball. In neither of them was there any proof of inherited syphilis, but in one the paralysis passed away under treatment by specifics in a manner which was very suggestive.

CASE 10.—A female infant, *æt.* 9 months, under care in 1876, at the London Hospital, had symmetrical ptosis and almost complete paralysis of all the ocular muscles. The lids drooped so as almost to cover the eyes. It was very difficult in so young a child to test each muscle, but so far as could be ascertained the paralysis was almost absolute, with the exception that the left eye moved a little both outwards and inwards. The condition had existed for fourteen days. The child was pale and fretful, but showed no signs of syphilis. There was an elder child, *æt.* 3, also healthy, but both of them had suffered from “thrush.” Iodide of potassium and mercury were ordered, and at the end of four months all trace of the paralysis had disappeared, and the child was cheerful and in good health. Five months after treatment was laid aside the child was seen again, and continued quite well. Two months after this, however, it was taken ill again, wasted, had convulsions, and died, but no information of this illness was given to Mr. Tay until some time after the death.

CASE 11.—The subject of Mr. Tay's second case was a girl, *æt.* 3, named A—, under care in March, 1878. Both eyelids drooped considerably, and several of the recti on both sides were paralysed. With the left eye she could look outwards but not in other directions, and with the right inwards and outwards fairly, but not in other directions. The pupils were of medium size, and sluggish. The symptoms had been coming on for six weeks, and there was a history of a slight fall. The child was fretful, but there were no indications of syphilis. A boy two years older than the patient was quite healthy. Iodide was ordered. Death occurred on April 19th, 1878, but no details of the latest symptoms are forthcoming, nor was a post-mortem obtained.

CASE 12.—I have met with one instance of this malady which was unquestionably in connection with inherited syphilis.

Abraham F— is at present unable to use any of the recti

muscles excepting to a slight degree, and his eyes are almost fixed in a divergent position. He is quite blind, and both discs are in an advanced state of atrophy. He has been at different times during the last few years under many of the ophthalmic surgeons of London, and also under Dr. Hughlings Jackson, by whom he was sent to me. He is florid and healthy-looking, but with a suspicious forehead, and quite characteristic teeth.

At the age of 13 his sight began to fail, and squint was noticed, and from this time his symptoms steadily advanced. For two years and a half he has been, as he is now, totally blind, with fixed eyeballs, and slightly drooping lids. There has been no evidence of increasing disease of the centres, and he still walks well, and has perfect use of all his faculties. He sleeps and eats well, and goes regularly to a blind school, where he reads by touch. He has now no headache, but in the earlier stages he had much headache and occasional "shiverings." His age is now 16.

His right eye is usually on a lower level than the left. He can only by great difficulty lift his lids so as to expose the whole of the cornea, and when at rest the corneæ are half covered, and he looks as if just going to sleep. Both eyes diverge. Both inferior recti are wholly paralysed, and both superior almost wholly so, the left being rather better than the right. The internal rectus of the right side is quite paralysed, and that of the left nearly so. The external rectus of the right is very feeble, but that of the left still acts moderately, being much the most active muscle on either side. All the obliques are paralysed.

CASE 13.—Symmetrical and almost complete ophthalmoplegia externa without implication of the iris or ciliary muscle, but with defect of all muscles supplied by the fifth, and, in slight degree, of those by the seventh. No history of syphilis.

A young man named George S— came to Moorfields in October, 1877, and was admitted under Mr. Waren Tay's care. He showed no signs of inherited syphilis. He was only 19, and entirely denied any history of acquired taint.

He was from Devonshire and of healthy family. In addition to investigations by Mr. Tay and myself, his symptoms were carefully studied by Dr. Hughlings Jackson, Dr. Barlow, and others. On both sides his eyelids drooped so as to almost cover the eye, and all the muscles of the eyeballs were exceedingly feeble. His pupils, however, acted well, and accommodation was perfect (entire absence of ophthalmoplegia interna, and probable integrity of the lenticular ganglion). The ocular muscles were somewhat more completely paralysed on the right side than the left, and the lid drooped more, but on both sides the degree of weakness was almost complete, and involved all the muscles. In addition to the muscles of the eye, all those supplied by the fifth nerve were on both sides defective, but their paralysis was not nearly complete, and on both the muscles supplied by the facial were also slightly weak. There was no defect of sensation, nor any symptoms referable to the spinal cord. The vision and other special senses were perfect, and the man appeared to be in good general health. There had never been much headache.

The symptoms above mentioned had been present six months, and were supposed to be consequent on a trivial blow from a piece of wood on the left temple. There had been an interval of three weeks between the blow and the first drooping of the eyelids, and the blow itself was so slight that it may be reasonably doubted whether it had any connection with the paralysis.

Through the kindness of Mr. Fernie, of Barnstaple, under whose care the man is, I am enabled to state that at the present date, fifteen months since he was under care at Moorfields, the symptoms remain without advance, but with some improvement. Treatment by iodide of potassium has been pursued, but not regularly, nor in large doses.

I may suitably place in juxtaposition with this case one in which, as in it, the fifth-nerve muscles were involved in association with those of the eyeball. It differs, however, definitely from the preceding one in that there can be no doubt that the patient is the subject of syphilitic taint. It is a very

remarkable case, but I must state its facts as briefly as possible.

CASE 14.—Madame de T— æt. about 30 years, and in good bodily health. She has notched teeth, and has suffered a severe attack of interstitial keratitis. A sister, like herself, is the subject of inherited syphilis. Before, however, we confidently attribute her nerve-symptoms to inherited taint, I am bound to state that her husband believes that she has had acquired disease as well. About this no certainty is attainable. She is at present the subject of paralysis of all the ocular muscles on the left side, with only slight weakness of these on the right, but with double paralysis of the fifth, both motor and sensory, and some defect of both facials. She was formerly hemiplegic in her right limbs, but this has passed off. The account of the development of her symptoms is obscure, but the paralysis of the cranial nerves preceded the “fit.” She has already derived much benefit from treatment by large doses of iodide of potassium. The non-symmetry of the ophthalmoplegia, and the occurrence of hemiplegia, separate this case somewhat from the group under our consideration.

I have seen double paralysis of the ocular muscles once—and only once—in association with a history of apoplexy, and the case, although probably not very closely cognate to the others, must be briefly mentioned.

CASE 15.—Abraham B—, a healthy-looking old man, æt. 72, came under care in 1874, having, on the right side, paralysis of all the muscles of the eyeball, except the internal rectus. The condition was symmetrical, but on the left side the degree of paralysis was not so great. The eyeballs were converged, so as to be almost buried in the inner canthi. Accommodation was lost, but vision almost perfect. He was partially hemiplegic in the left limbs, and the muscles supplied by the right facial were weak. The history was, that the symptoms had followed a fit which had occurred suddenly three or four years ago. There was no history of syphilis, but its possibility was not denied. His vision failed

during the period that he was under observation, but in other respects his symptoms were stationary. It is difficult on any hypothesis of hæmorrhagic disorganisation to explain such a group of symptoms, and it is, after all, quite possible that the apoplexy was an accidental concomitant, and that the ophthalmoplegia was of the same nature as in the other cases.

P.S.—Since my paper was sent in I have found in my note-books two cases of this affection which I have omitted to mention.

CASE 16.—The subject of the first is a married lady whom I attended with Dr. Hughlings Jackson some years ago. She had double ptosis, with incomplete paralysis of most of the ocular muscles, fixed pupils, and loss of accommodation. She had been liable, previously, to most violent attacks of vomiting ("abdominal crises"). She improved very much under specifics. She became subsequently hemiplegic. She is still living (ten years after the first symptoms), but is confined to her room, and many of her ocular muscles still paralysed. There is much reason to suspect syphilis.

CASE 17.—The subject of the second is a sailor, who had syphilis six years ago, and in whom both-sided paralysis of the eye-muscles with ptosis set in rather suddenly after two months' premonitory symptoms in the form of failing accommodation.

I have detailed notes of both these cases, but must not further trespass on the patience of the Society. It will be seen that these add two more to the list of those in whom the disease appears to be due to syphilis.

Comments on the Series.

The facts are too few to permit of statistical analysis, but a brief summary may, perhaps, be useful. Of the fifteen cases four only occurred in females, and two of these were young children. One of the males was a boy the subject of inherited taint, and the remaining ten were all adults. In eight of the fifteen it seemed certain that syphilis was the

cause, in six acquired, and in two inherited. Of the remaining seven it may be said that a reasonable suspicion of syphilis might be entertained in several. Had it not been for an almost accidental revelation of the truth in the case of the man S— after prolonged fruitless investigation, I should certainly have asked the Society to believe that his was an example of the disease without any probability of syphilis. With such a fact in mind, one feels that it is almost impossible to make the negative even fairly probable. In Case 9 no direct question was asked, the patient being a single woman. In the two young children, although nothing could be proved, there were some suspicious facts, and one recovered under the iodide. The case in which evidence is most conspicuously absent is No. 13, but in this the patient is a young man, who may have denied the true history, or who may have either inherited a taint or had the acquired disease in some irregular way, of which he knew nothing. On the whole, the evidence which connects this affection with syphilis is exceedingly strong, and that which favours the belief that it can occur independently of it, must be held to be open to some doubt.

In five of the patients (all but one, known to be subjects of syphilis), the optic nerves were affected, and blindness, with white atrophy, resulted. In two cases the fifth nerves were symmetrically affected, and in two there was slight affection of the facials. The almost constant escape of the facials must be held to be a remarkable fact. In one case the palate was affected, and in one, the same patient, smell was lost. None of the patients were deaf and none had lost taste, and in only one was there any material anæsthesia of the skin of the face. In six cases the lower extremities were more or less weak, and liable to pain, the condition approaching more or less closely to locomotor ataxy. I much regret that, owing to imperfect knowledge on my part at the time of their occurrence, the details of facts in reference to this disease are often incomplete. There can, however, be no doubt that ophthalmoplegia externa is sometimes a part of the general malady known as progressive locomotor ataxy,

especially when that disease is due to syphilis. One of the patients became insane, and another was before death liable to attacks of violent mental excitement. Four of the fifteen are known to be dead, but of these a post mortem was obtained in only one. In several cases, owing either to the blindness or the youth of the patient, it was impossible to estimate the state of the accommodation, but in a certain number it was proved to be perfect, in a few it was absent, and in a few it was impaired. The pupil, never contracted, was almost always sluggish and of medium dilatation. In one well-marked case it acted fairly. In no single case was it very widely dilated. From these facts we may infer that the lenticular ganglion is often free from disease, and that the vaso-motor filaments, although often enfeebled, are not usually paralysed.

It is difficult to make any confident statements as to the progressive tendencies of the malady of which external ophthalmoplegia is a symptom. It is very definitely influenced for good by treatment, and in nearly every case specific measures were adopted. We may conjecture, however, from what happened in several, that in most instances it is an aggressive malady, and would end fatally if treatment were not resorted to. The effects of remedies in several of the cases were very remarkable, the patient being rescued from a very dangerous condition. At the time that most of the cases were under treatment my opinions, as regards the nature of the malady, were far less clear than at present, and hence a hesitancy in treatment, which was probably often prejudicial. The patients were benefited up to a certain point, but relapses occurred, and the remedy was not pushed sufficiently. It would seem that iodide of potassium is by far the best means of treatment, and that it ought to be given over very long periods, and in increasing doses without any limit as to precise quantity, excepting its effect on the symptoms. Although relapses are common, yet in several of the cases narrated a complete arrest appears to have occurred, no treatment having been resorted to for several years. In none, however, was the recovery complete.

NARRATIVE
OF A
CASE OF TRUE LEPROSY,
IN WHICH
COMPLETE RECOVERY HAS TAKEN PLACE.

BY
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SENIOR SURGEON TO THE LONDON HOSPITAL AND TO THE BLACKFRIARS
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(Received December 10th, 1878—Read February 11th, 1879.)

THE subject of the appended narrative has been under my observation at different times during the past twenty-seven years. When I first saw her she was suffering from leprosy in a severe and well-characterised form. It was the mixed kind, partly tubercular and partly anæsthetic. She has now regained good health, and although in the state of her right hand, in which ulnar paralysis exists, in the dusky tint of her skin, and in its loss of sensibility in various parts, we have permanent consequences of the disease, yet there have not been for many years any indications of tendency to relapse. All tubercles have long since disappeared, and both as regards skin and nerves, we may consider that reparation has taken place as far as it was possible. As an instance of proved recovery from a disease which is usually steadily aggressive, and especially because I am able to produce the

patient for inspection, I think the case not unworthy of the attention of the Society.

It will be seen that the disease was acquired by an immigrant into a leprosy district who did not inherit any proclivity, and that the recovery took place after a permanent return to England. The patient, during her residence abroad, had been exposed to no hardships, and her case seems to me to lend some support to the opinion which I have long held that the malady is due solely to diet, and that its special cause is probably fish.

Case of recovery from severe leprosy acquired twenty-eight years ago in Jamaica.—Mrs. Harriet I—, æt. 71, was born in Portsmouth, of Jewish parents who had been both born in England. When thirty-two years of age she went to the West Indies, being in excellent health at the time. She lived at Montigua Bay, Jamaica, eleven years, and during the greater part of that time enjoyed good health. Her diet was beef, turtle in all forms (at sixpence the pound), fish and vegetables. The first was plentiful and good. She thinks she took fish almost every day, but adds, "I never took any fish that would injure me—barracouta, or anything like that." About a year before she returned to England she began to suffer from leprosy. The first local ailment that she remembers was a swelling on the right little finger and within a month or two blotches came on her forehead. It was diagnosed on the spot as "something of the nature of leprosy," and she was advised to return to England.

On arriving in England (æt. 44) she at once presented herself at the Hospital for Skin Diseases, where, under Mr. Startin's care, I often saw her. We took great interest in her case as a well-marked example of true leprosy. Her face was covered with dusky tuberculous folds of thickened skin. Her arms also showed many patches, and were in parts anæsthetic. She got no benefit from our treatment, and after ten months' attendance left us, and obtained admission into Guy's Hospital under the care of Dr. Addison, who, she says, wished to have a wax model made.

She remained in Guy's Hospital three months, where, she believed, an attempt was made to salivate her, but without success. She next went under Mr. Hunt's care, who pushed a course of arsenic, and, she thinks, made her eyes sore. After this she was under care at Moorfields (Mr. McMurdo) for her right eye, and has continued to attend there almost ever since.

As regards the cure of her skin she states that all drug treatment had seemed to fail and she had left it off. About two years after her return from Jamaica, the skin being still much in the same condition, the return home of a wealthy relative placed good wine within her reach, and she took freely of port and champagne, under which rapid improvement ensued, and in about a year she thought herself well. She has had no return in the skin, and "has never had a day's ill health since; not to speak of."

Mrs. I— is now a very young-looking woman for her years (71), florid, and healthy. She still shows various conditions which have resulted from the leprosy. Her skin, although nowhere quite anæsthetic, excepting in the region of the right ulnar nerve, is in many parts deficient in sensation. Her right eye is inflamed and the cornea ulcerated, with deposit in its structure and much persistent conjunctival congestion. The state of her eye is that characteristic of leprosy, but there have not for many years been any aggressive changes.

As regards the frequency of recovery from conditions of advanced leprosy, it is probable that they are infrequent, and that they occur only under conditions, such as those present in this instance, of complete change of place of residence. I have never myself witnessed a similar case, nor do I know of any which have been authenticated in English practice. In former times many Norwegians suffering from leprosy emigrated into the United States, and I believe that it was thought that a certain number recovered, but I am not aware that this fact has ever been proved by the citation of individual cases. It is, indeed, not often that a patient remains sufficiently long under the observation of the same

surgeon for him to be able to speak definitely of the first and last state of such a protracted malady. I have seen during the last twenty years probably about a dozen cases of leprosy in Europeans. Two of them I know to be dead, but the others have passed from my observation. In every instance I have forbidden the use of fish as the first essential in treatment.

ON THE
DIAGNOSIS AND TREATMENT OF
RUPTURED BLADDER.

BY
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HAVING recently had under my care a case of rupture of the bladder into the peritoneum, in which I opened the abdomen and closed the aperture in the bladder with stitches, with a fatal result, I have been led to look closely into the history of recorded cases of ruptured bladder with the following results.

The symptoms of ruptured bladder are often not so marked as might be expected from the classical descriptions of text-books. A man with a full bladder receiving a blow upon the belly, may no doubt be conscious of something giving way and become faint; but where a severe injury, such as a "buffer accident," has been inflicted upon the pelvis, it is not uncommon to find, *post-mortem*, a rupture of the bladder which has given rise to no special abdominal symptoms during the last few hours of life. Indeed, so equivocal often are the symptoms that the reputed cases of recovery after rupture of the bladder have formed grounds for disputation between able surgeons as to whether or not the viscus was really ruptured.

In my own case the patient was under medical care for an entire day without the accident being recognised, and on admission to the hospital his case was regarded as one of injury to the kidney rather than the bladder, until my visit a few hours later. Indeed, had my patient recovered without operative interference, I believe he might have been said to have escaped injury to the bladder, as in Mr. Chaldecott's and Dr. Thorp's cases.

For the following detailed report of my case I am indebted to Mr. Samuel Burton, late Surgical Registrar of University College Hospital.

Jesse B—, æt. 47, a labourer, was admitted into University College Hospital, 11 a.m., November 25th, 1878, with the following history:—At 9.30 p.m., November 23rd, after patient had drunk two pints of beer, he began "larking about" with his companions, one of whom threw him violently on his back by putting his leg behind patient and forcing him backwards by his elbow, which hit him "in the stomach." He suffered great pain after the fall, and was taken home, where he was attended by a doctor who sent the following account with him:—"Patient is suffering from hæmaturia and retention of urine. A No. 6 catheter has been passed at intervals, but only a few ounces of bloody urine have been drawn off, the bladder remaining distended. He has been catheterised this morning (November 25th) with the same result as before. Opium has been given since November 24th, a.m., and patient appears better." Patient is quite sure he passed water freely half an hour before the accident.

Condition on admission.—Patient looks very ill, is extremely anæmic, has an anxious expression of countenance, is breathing quickly, mainly thoracic. Skin is moist. Pulse feeble and rapid. Pupils contracted from opium. Patient is lying on his right side with his knees drawn up, complaining of great pain in the lower part of the belly.

Examination of abdomen.—The skin over the front of the abdomen is reddened by a sinapism. The abdomen is pro-

minent in front, no bulging of the flanks ; it is firm and tense to the feel, and very painful on the slightest pressure, notably over the hypogastrium. There is dulness above the pubes to a point midway between pubes and umbilicus. The left flank is dull on percussion, the right resonant ; on turning patient over to right side, the left flank becomes resonant. There is no bruising of the back, but there is tenderness in the left lumbar region.

A No. 7 catheter was passed (this was the largest size that could be passed, owing to a stricture three inches from meatus) and three ounces of bloody urine drawn off, which came in an intermittent stream. No more urine coming away, the catheter was withdrawn, and the eyes were found to be plugged by a clot of blood. There is no pain on pressure over perinæum, and the pubic symphysis and arches are intact.

At his visit Mr. Heath saw the patient at 3 p.m., and diagnosed ruptured bladder. Patient having consented to undergo an operation he was taken into the theatre at 4 p.m., and ether was administered.

Mr. Heath first of all passed a No. 7 catheter, and found that bloody urine was ejected by intermittent, irregular jerks, corresponding to expiration. About half a pint was withdrawn. The catheter passed a long way, but was not felt to go through any rent in the bladder ; it was freely movable, as if in some large cavity. The pubes being shaved and washed with carbolic lotion, an incision was made in the middle line just above the pubes for two inches, and the tissues divided down to the peritoneum, which appeared blue, the recti muscles which were firmly contracted being held aside by retractors with difficulty. The peritoneum was then picked up and a cut made into it, when a gush of fluid like that drawn off by the catheter came out ; a large quantity of clots was then taken out from the peritoneal cavity. Mr. Heath having introduced his finger into the peritoneal cavity, found a long rent in the posterior wall of the bladder high up. It was proved to be a rent in the bladder, by passing a catheter through it from the urethra. The rent was then sewn up by

a continuous catgut suture firmly tied at both ends. The clots were removed as far as possible from the peritoneum and the cavity sponged out after injection with warm water, and a long large-sized drainage-tube was inserted at the lower angle of the wound, the upper part of the wound being brought together by deep and superficial sutures. The carbolic spray ceased working before the operation was completed. A catheter was passed into the bladder, to which was afterwards attached some india rubber tubing leading into a vessel under the bed. Patient being put back to bed, a hot poultice was applied to the abdomen, and opium was administered, gr. j in pil. 4 tis horis.

At 11 p.m. patient expressed himself as much better. His anxious Hippocratic aspect had passed off; pulse had improved; no sickness; abdominal pain much less; distension relieved.

November 26th, 10 a.m.—Patient has passed a quiet night, almost free from pain. He lies with legs outstretched.

Urine.—The amount that has drained away since the operation is $28\frac{1}{2}$ ounces; it is apparently free from blood, acid, sp. gr. 1018, contains one-twelfth albumen, colour brownish-yellow, somewhat turbid; under the microscope some red blood discs are seen. Temp. $99\cdot2^{\circ}$; pulse 112, moderately full, and less compressible.

Examination of abdomen.—The prominence and distension are much less marked, and there is but little pain on pressure. The bowels have not acted, but flatus has been passed per anum. The drainage-tube was removed, a little bloody fluid being expressed from the wound. Poultices continued. He has taken three eggs and brandy 3ij every hour.

November 27th, 10 a.m.—Patient has passed a quiet night, sleeping for the greater part of the time. He is dozing now, and lies with legs outstretched. Temp. $98\cdot2^{\circ}$; pulse 112; no sickness. Since 9 p.m. last night patient has passed four copious stools.

Urine.— $44\frac{1}{2}$ ounces passed in the last twenty-four hours,

besides some into the bed, of normal colour, acid, and free from blood.

The abdomen is a little more distended, but not painful, except at the epigastrium. There is but little discharge from the wound, only sufficient to soak a small piece of oakum placed over the wound, and which is changed every four hours.

November 28th, 10 a.m.—Patient has slept well through the night, complains of feeling “blown up with wind.” No sickness or hiccough. Tongue moist, coated with a thick, yellow fur. Temp. 98° ; pulse 100. The abdomen is very tense but not painful, the left flank is dull. A considerable quantity of fluid has drained from the wound, the edges of which are sloughy.

Urine.—92 ounces passed in the last twenty-four hours, all but the last four ounces of normal colour, contains no albumen, and is free from blood. These four ounces are of a bright red colour, slightly acid. Sp. gr. 1020, one-twelfth albumen.

November 29th, 10 a.m.—Patient passed a very restless night. He has had constant vomiting and can keep nothing down. Has passed several motions into the bed. He is looking very ill this morning, expression anxious, and features pinched. Pulse small and indistinct; temp. 98.4° .

Abdomen is very distended and somewhat painful. The wound looks very sloughy, and there has been scarcely any discharge of sero-purulent fluid.

Urine.—45½ ounces passed of a chocolate colour, nearly solid, with albumen; neutral. Sp. gr. 1018.

4 p.m.—Patient is much worse. Sickness and diarrhoea continue.

10 p.m.—Patient is sinking fast.

11 p.m.—Died, six days after the accident.

November 30th.—Post-mortem made at 2 p.m., fifteen hours after death, weather cold, rigor mortis well marked.

The abdomen being opened by a crucial incision, the small intestines were seen considerably distended. For two inches around the wound in the abdominal wall the intestines were adherent by recent lymph to each other, and to the abdo-

minal parietes. Above and on each side of these adhesions there was no trace of peritonitis. On tearing away these adhesions, some coils of intestine were seen lying over the pelvis, glued together and to adjacent parts by recent blood-stained lymph. On lifting these coils upwards, the recto-vesical pouch of peritoneum was exposed, containing about six ounces of clotted blood, black in colour, and moderately offensive in odour. The rent in the bladder was also visible, the lower part of the wound gaping. Some blood lay over the cæcum and the ascending colon; this had probably overflowed from the pelvis.

The *thorax* was then opened. *Heart and lungs* apparently quite healthy. *Liver* large and fatty, weight 4 pounds 2 ounces. *Spleen* normal. *Intestines* removed, no sign of enteritis along the whole tract.

Kidneys and pelvic organs removed *en masse*. There is no sign of pelvic cellulitis or of extra-peritoneal hæmorrhage. No injury to the large vessels of the abdomen or pelvis. No fracture of the symphysis pubis, or any of the pelvic bones.

Examination of bladder.—There is a rent in the mid-line of the posterior wall of the bladder 2 inches in length, extending upwards as high as the apex. The lower third of the rent was gaping, the edges of the rest were approximated by the catgut suture, the lower end of which was free and loose. The bladder was slit up in the middle line in front, and on each side close to the prostate; the mucous membrane was blood-stained.

The recto-vesical pouch was lined by tolerably firm, blood-stained, adherent lymph.

Kidneys are of normal size, capsules smooth and strip off readily. *Pelves and ureters* healthy.

Brain and its membranes healthy.

My reasons for believing that the bladder was ruptured were:—1, the pallid pinched appearance of the face betokening some serious visceral lesion; 2, the distended condition of the abdomen; 3, the fact that a small quantity of clear urine was drawn by the catheter immediately on reach-

ing the bladder, and on further introduction a quantity of bloody fluid presumably from the peritoneal cavity; 4, that warm water injected through the catheter was distinctly felt by the patient in the groins and abdomen. The passage of fluid from the peritoneal cavity became more apparent when the patient was breathing heavily under ether, for then the fluid flowed and stopped with each respiration. This symptom was noticed also by Mr. Willett in his case, 'St. Bartholomew Hospital Reports,' 1876, and I am inclined to lay stress upon it, since so long as the muscular coats of the bladder are entire, I believe micturition never is so directly influenced by the respiration.

My reasons for selecting the proceeding I did were, 1, the hopelessness of the case if left untreated; 2, the fact that the operation had been recommended by Mr. Holmes in his 'Principles and Practice of Surgery,' although I was not aware of its having been performed. I have since found that Mr. Willett performed the operation at St. Bartholomew's Hospital in 1876, using interrupted silk sutures, the patient dying the next day. Mr. Willett's case in many respects resembled mine, but a somewhat lesser time had elapsed between the accident and the operation. I can fully confirm Mr. Willett's remarks as to the difficulty of the operation, due to the muscular condition of the abdominal walls; but I used a smaller incision than he did, and consequently did not allow of any escape of the intestines.

I must confess my surprise at the very large quantity of blood effused into the peritoneum by a rupture of a not highly vascular organ such as the bladder. On opening the abdomen I removed a quantity of clot, and subsequently washed out more, but after death a considerable quantity was found in the recto-vesical pouch. Rather against my own judgment, at the time of the operation I introduced a large india-rubber perforated drainage-tube into the pelvis, and secured it at the lower end of the wound, and I find that Mr. Willett adopted the same practice. Finding that nothing came through the tube, but that clear serous fluid from the peritoneum welled out by the side, I withdrew it on the

second day, and I cannot but think that its presence was harmful to the patient. The peritonitis was limited to the pelvis, and its greatest intensity had been exactly where the tube passed. Had I introduced a tube through the recto-vesical pouch of peritoneum and brought it out at the anus I should have established a dependent opening, and been able to wash out the blood which collected there. Tapping the recto-vesical pouch from the rectum in cases of ruptured bladder was suggested by Dr. Harrison in his well-known paper in the 'Dublin Journal of Medical Science,' 1836, but I am not aware of its having been performed.

Although *post-mortem* the lower part of the tear in the bladder was found open from the slipping of the knot or giving way of the catgut, I think it was completely closed at the operation, since clear urine in large quantity flowed through the catheter up to the fifth day, when bloody urine was noted in the evening, and the whole of the urine passed on the last day contained blood. I may say, that the first stitch was put in at the lower end of the opening by means of a needle set at right angles to the handle, and was then firmly tied. One end of the catgut being then used by an assistant to pull the bladder up out of the pelvis, I threaded the other end into an ordinary needle, and carefully sewed the opening up with a continuous suture, a great part of which is still visible in the preparation.

Putting aside the few undoubted cases of recovery from rupture of the bladder outside the peritoneum, we come to the cases of alleged recovery after rupture into the peritoneum, about which much controversy has arisen, and mainly because those surgeons who regard rupture of the bladder as a necessarily fatal accident, refuse to believe in a recovery without a demonstration of the lesion, which is of course impracticable. A careful consideration of Mr. Chaldecott's case, 'Provincial Medical and Surgical Journal, 1846,' leaves no doubt in my mind that it was one of rupture into the peritoneum, and Mr. Aston Key seems to have fully endorsed Mr. Chaldecott's view. The patient was treated by catheterism and opium, and suffered from peri-

tonitis, and gout presumably due to absorption of urine from the peritoneal cavity. It is of course possible that the urine may have been shut off by adhesions from the general cavity of the peritoneum, but it is remarkable that on the sixth day after the accident the patient having attempted to micturate "felt something give way, and a burning pain all over his stomach and bowels, as if boiling water had been poured over them, and the same symptoms of faintness and distress as when the accident first happened."

Dr. Gross, of Philadelphia, in his work entitled, "Practical Treatise on the Diseases and Injuries of the Urinary Bladder," &c., published in 1851, proposed abdominal section for the evacuation of the urine in these cases, and Dr. Walter has reported in the 'Philadelphia Medical and Surgical Reporter,' February, 1862 (quoted in Ranking's 'Abstract,' 1862, vol. ii), the case of a man, aged 26, in whom, ten hours after the accident, an incision was made in the linea alba, and a sponge introduced into the peritoneal cavity. A pint of urine and blood was thus mopped out, and a rent two inches long was observed in the base of the bladder. The bladder was left to itself and the catheter used, but apparently not tied in. The patient made a complete recovery.

It should be mentioned, that both Bonet¹ and Cusack² had performed Paracentesis abdominis for the evacuation of urine, but with fatal results.

In the 'Dublin Quarterly Journal of Medical Science' for November, 1868, Dr. Henley Thorp reported, perhaps the most remarkable case of recovery after ruptured bladder on record. A farmer, aged 30, was thrown from his horse and was found insensible. When he became conscious "he experienced a severe pain at the bottom of his belly, attended with an urgent desire to pass water, but no power of emptying his bladder." Dr. Thorp found him sitting up in great distress, and passed a full-sized gum elastic catheter into the bladder. "At first no fluid escaped; but upon

¹ 'Sepulchretum Anat.,' lib. iii, sec. xxiv, obs. 12.

² 'Dublin Hosp. Reports,' 1818, vol. ii, p. 812.

pushing the instrument onwards, and at the same time turning it a little on its axis, about a tablespoonful of bloody urine flowed out. By changing the position of the patient from side to side, turning him over upon his knees, and substituting a silver for the gum elastic instrument, I at length succeeded (says Dr. Thorp) in obtaining nearly half a pint of urine mixed with blood." A few hours later (it is not stated how many after the accident) Dr. Thorp passed an elastic catheter into the bladder, when "its movements were at first restricted and painful, until after cautiously probing and turning its point, it entered nearly its full length, when a different feeling of resistance was communicated, and it could be moved about with somewhat greater freedom." A tablespoonful of reddish urine now escaped, and Dr. Thorp proceeded to inject warm water from a half-pint elastic bottle three several times, allowing each eight ounces to be retained for a couple of minutes, and then to return through the catheter. "At first the water returned of a reddish tinge, but the last half-pint was clear and bloodless." The catheter was now withdrawn partially, so that its end projected into the bladder, and when the urine began to come away was tied in. Forty leeches were applied to the abdomen followed by poultices, and 1 grain of opium with $\frac{1}{2}$ grain of calomel was given every two hours. The catheter was kept in for eleven days and the patient recovered, having had symptoms of sub-acute peritonitis more than once.

In 1863 Dr. Stephen Smith, of New York, suggested cutting into the bladder from the perinæum, as in lateral lithotomy, in order to give free exit to the urine in cases of ruptured bladder. The operation has apparently been done only twice, viz. by Dr. W. J. Walker, in a case of laceration of the front of the bladder outside the peritoneum; and secondly, by Dr. Erskine Mason ('New York Medical Journal,' 1872) in a case of presumed rupture into the peritoneum. Both patients recovered.

In Dr. Mason's case the operation was done two days after the accident, when symptoms of general peritonitis

had set in, and *per rectum* a tumour could be felt posterior to the prostate and to the left side. The bladder having been opened on a staff in the usual way, Dr. Mason felt confident that his finger detected a rent in the posterior wall of the bladder, but he did not examine the opening thoroughly, fearing to do injury. A quantity of bloody urine escaped through the incision, and the tumour felt *per rectum*—presumably the recto-vesical pouch distended with fluid—disappeared. In a fortnight the patient was convalescent and completely recovered.

Mr. Willett, who quotes Dr. Mason's case at some length, controverts his view as to the bladder having been ruptured into the peritoneum, though he allows that the operation saved the man's life. I am not aware that the operation has been performed in a similar case in this country; but Mr. Bryant, in his 'Manual of Surgery,' recommends the proceeding in cases of ruptured bladder.

To sum up the cases of recovery after reputed rupture into the peritoneum, they are four in number:

1. Mr. Chaldecott's case, treated by catheterism of the bladder and peritoneum.
2. Dr. Walter's case, treated by abdominal incision to evacuate the urine.
3. Dr. Thorp's case, treated by catheterism of the bladder and peritoneum, and washing out the peritoneum through the catheter.
4. Dr. Mason's case, treated by lateral lithotomy.

These cases alone are sufficient to encourage the surgeon not to abandon a case of ruptured bladder as utterly hopeless, and Dr. Thorp's seems to me the most satisfactory of them all. An abdominal section for mere evacuation of fluid seems to possess little advantage over paracentesis abdominis in the linea alba, and must be less effective as a drain than tapping the recto-vesical pouch *per rectum*. Having experienced the very great difficulties to be encountered in stitching up the bladder with plenty of able assistants, I cannot recommend the proceeding for adoption when there would be the best chance of success, viz. imme-

diately after the accident when the surgeon first sees his patient. I may say that I was well acquainted with Dr. Thorp's case, but was not aware of Mr. Willett's experience. Had I known that the trial of stitching up the bladder had been made without success, I believe I should have resorted to Dr. Thorp's expedient of washing out the peritoneum, which I would certainly recommend for imitation in future cases. It seems to me to offer as good, if not better chances of success than any other proceeding, and has the great advantage that it can be put in action promptly, which is after all the great point. In addition it introduces no new element of danger to the patient, nor any serious surgical proceeding which may be distasteful to his friends. The operation of lateral lithotomy might, I think, be fairly reserved for cases of ruptured bladder in which the catheter could not be passed into the peritoneum from the urethra.

REMARKS
ON
ONE HUNDRED AND FIFTY OPERATIONS
FOR EXTRACTION OF CATARACT.

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IN the table which accompanies this paper I have put down the leading facts with the results of a hundred and fifty extractions of cataract performed on a hundred and thirty patients; in the table two numbers bracketed together signify two eyes of the same individual. Seventy-one of the patients were males and fifty-nine were females.

The results are comprised under three heads: successful, partially successful, and failure. Under the first are placed all eyes that could read print varying in size from $D = 0.5$, to $D = 4$ of Snellen's test types at a distance of from 20 cm. to 50 cm., and had vision for distance $= \frac{6}{8}$ to $\frac{6}{60}$, or could tell the time on a watch within a fortnight or three weeks of the operation, aided in each case by a suitable convex lens. As far as No. 112 in the table the old edition of Snellen's types and lenses numbered by focal lengths were used, in

those after No. 112 the new edition of types and metrical lenses.

Under the second head come eyes which could count fingers and distinguish one from the other; thus, tell the thumb from the fingers, the small from the forefinger, also whether the front or back of the hand was looked at, or had vision enough to enable the patient to go about without a guide.

Under the third head are placed those eyes which saw no better, or worse, than before the operation.

The number of eyes in which the operation was successful is one hundred and fifteen, 76·6 per cent of the whole; partially successful, twenty-four, 16 per cent; failures, eleven, 7·3 per cent. So that in 92·6 per cent the patients were the better for the operation.

The ages of the patients varied between three and eighty years. One hundred and twenty-four of the cataracts were nuclear, twenty-six cortical. By nuclear I mean that form of cataract met with in persons past the middle of life, in which the nucleus or central part of the lens is opaque and hard, the cortical layers being opaque but rather softened, the opacity of the nucleus being often first in point of time, frequently described as senile or hard cataract.

By cortical I mean the form met with in young people, and at all ages from injury, in which the opacity commences in the cortical layers of the lens, and subsequently invades the nucleus, the whole being of a consistency varying between a gelatinous and completely fluid condition, often described as juvenile or soft cataract.

The methods of extraction were three—small flap section upwards, or downwards, associated with iridectomy, one hundred and four; linear section upwards, or downwards, also associated with iridectomy, twenty-five; oblique corneal section downwards, as a rule without iridectomy, twenty-one. In the two first the iridectomy was, as a rule, performed at the time of extraction, but in several cases as a preliminary some weeks or months previously.

In six cases a traction instrument, scoop, or sharp hook

was required to remove the lens. In five vitreous was lost. Secondary operations—iridectomy, or tearing through opaque capsule—were required in twenty-nine (19·3 per cent). Iritis, sufficient to cause impairment of vision, occurred in twenty-four cases (16 per cent.). Anæsthetics were given in one hundred and twenty-eight, the operation was performed without in twenty-two cases.

The number of cases is too small to enable me to draw comparisons between different methods of operating, and I do not pin my faith exclusively to any one, but my impression is that the extraction by small flap section with iridectomy is that which is best suited to the majority of cases.

The operation with upward section is performed as follows:—The patient is placed in the supine position, brought thoroughly under the influence of an anæsthetic, the lids held open by a wire speculum, the globe fixed by holding the conjunctiva and subconjunctival fascia near the lower corneal margin with the fixing forceps, and drawn gently downwards. A Graefe's knife is then entered in the sclero-corneal junction on the outer side of the globe at a point midway between the upper margin of a moderately contracted pupil and the upper border of the cornea; the surfaces of its blade being kept parallel to the plane of the iris, it is pushed gently and slowly across the anterior chamber, and its point brought out at a spot on the inner side of the globe corresponding to that of entrance on the outer; then by a gradual sawing movement the knife is made to cut its way out upwards, the line of section corresponding as nearly as possible to the sclero-corneal junction, encroaching on neither cornea nor sclera; thus a flap comprising about one third of the cornea is formed. Iridectomy is then performed (if it has not been already done as a preliminary), but the piece of iris removed need not be large. My object is to make a pupil which resembles as nearly as possible an ordinary key-hole in form; should the eye turn upwards it must be drawn down by an assistant during the removal of the piece of iris, but I never let any one else

touch the eye if I can possibly avoid it. The next step in the operation is the laceration of the lens capsule. For this purpose I use a small sharp hook with flexible stem, which can be bent to any required angle, and which is gilded, so that it can be seen when in the anterior chamber much more distinctly than white metal or steel. A few scratches with the hook tear up the capsule most satisfactorily.

The cataract is removed by gently pressing with the curette upon the globe, which is drawn downwards if necessary, just below the inferior margin of the cornea. The bulk of the lens having escaped care is taken by gently pressing on the cornea from all sides towards the centre to get any soft cortical matter which may remain behind the iris into the pupil, and then gently press it out through the incision. So soon as the pupil appears black and clear the speculum is removed and the eye carefully closed, two small folds of lint wetted in cold water are then placed upon the lids of both eyes, and the whole secured by a bandage which has been made by Mr. Dunnage, surgeon to the Central London Ophthalmic Hospital.

In extraction by linear section the details of the operation differ from the small flap only in the form and position of the incision. Puncture and counter-puncture are made in the sclerotic beyond the sclero-corneal margin, at points in a line rather below the upper margin of the cornea. The Graefe's knife is entered and passed across the anterior chamber with the surfaces of its blade parallel to the plane of the iris, but as soon as its point has emerged on the inner side of the globe its edge is turned straight forwards and made to cut its way out just within the sclero-corneal margin.

In extraction by oblique corneal section no speculum or fixing forceps are used, but the lids are held open, and the globe steadied by the fingers. The section is made downwards by transfixing the globe with a Graefe's knife through the sclero-corneal margin, the puncture and counter-puncture being in a line rather below the centre of the pupil.

The edge of the knife during its introduction and passage across the anterior chamber is directed forwards so that the surface of the blade forms an angle of about 45° with the plane of the iris. As soon as its point has fairly emerged on the inner side of the globe it is made to cut its way out, by a gradual sawing movement, through the cornea, about midway between the lower border of the pupil and the sclero-corneal margin. As a rule no iridectomy is performed, but in cases where the pupil does not readily dilate it is well to remove a small portion of iris from its margin; the capsule is lacerated with the sharp hook, the cataract removed by making pressure with the forefinger of one hand upon the upper lid drawn up just enough to clear the upper margin of the cornea, aided by pressure similarly applied below with a finger of the other hand upon the lower lid. Any soft matter that may remain after the bulk of the cataract has escaped is carefully pressed out, the iris, if it has prolapsed, returned with the curette, and both eyes bandaged as after the small flap extraction.

The small flap section has, in my opinion, the following advantages:—The wound is made in a portion of the eyeball the vascular supply of which renders its healing power very great, and the flap being small the chance of suppuration is but slight. The risk of loss of vitreous at the time of the operation, or of choroido-iritis subsequently, is less than in linear section. The alteration in curvature of the cornea is not so great as after oblique corneal section.

With regard to the relative advantages of upward and downward sections, so far as vision is concerned, that for reading or near work is equally good in either; distant vision, however, is not quite as good after the latter as the former. In some cases where both eyes have been operated on in the same patient I have done one with upward, the other with downward section, and the former gave the best vision for distance, while for reading sight was equally good in both. The reason of this is, firstly, that from the size of the pupil, which instead of being to a great extent covered by the upper lid, lies, when the eyes are directed straightforwards,

entirely in the palpebral aperture, too much light enters the eye and causes confusion. Secondly, the patient looks through a portion of the cornea which may have become irregular from the cicatrix of the wound.

Other disadvantages of downward section are:—The wound is not so well covered by the upper lid as in upward section, and from its position is liable to be irritated by the lower lashes should entropion occur. If closure of the pupil or opacity of the cornea take place there is a difficulty in making an artificial pupil, as the greater part of the normal cornea and iris lie beneath the upper lid, by which the new pupil would be covered. The downward section, however, has this great advantage, it renders the operation of extraction extremely simple and easy, and lessens to an immense extent the risk of accidents during its performance. Thus, there is no necessity for the use of a speculum, or if one is used it can be removed so soon as the section is completed; it is never necessary to draw the eyeball downwards when performing the iridectomy, or, indeed, to touch it with fixing forceps after completion of the section. The cataract can be removed by pressure with the fingers upon the partially closed lids instead of by an instrument applied directly to the eyeball as in upward section; and, lastly, should an accident happen, such as escape of vitreous or displacement of the cataract necessitating the introduction of a traction instrument, the section is in such a position that the requisite manipulations can be carried out without touching the eyeball with forceps.

My practice is in all cases where I expect any difficulty, or operate without anæsthesia, to make my section downwards, as a rule employing no speculum or fixing forceps, but holding the lids open and steadying the globe with my fingers. In plain straightforward cases, and with anæsthesia, I make my section upwards, keeping the lids open by a speculum, and fixing the globe with forceps.

With regard to the performance of iridectomy I rarely extract without it. Some of my cases of oblique corneal section gave brilliant results, but such are the exception,

the rule being that a prolapse of iris takes place which most probably renders an iridectomy necessary at some time or other, and at least causes a considerable amount of irritation and leaves an anterior synechia, but may even set up glaucomatous change and destroy the eye.

The greater part of my experience of extraction without iridectomy has been gained from the practice of others; I have, however, seen enough of it to determine me to perform the operation but rarely, although the cases in which I have done it were, on the whole, very satisfactory.

My rule is in cases of mature cataract to perform iridectomy at the time of extraction, in immature as a preliminary as long before the lens is removed as possible, and in cases where the nucleus of the lens alone is opaque I make the artificial pupil downwards, as in this position it considerably improves vision so long as the margin of the lens remains clear.

In the selection of cases for operation I keep in view only two points; 1st, that the perception of light is good; 2nd, that the greater part of the lens is opaque. In several of the cases contained in the table the cataract was associated with disease of the deeper structures of the globe, chiefly the choroid; such form a large proportion of the partially successful cases, the remainder being accounted for by dense capsular obstruction, iritis, changes in the vitreous and choroid after extraction.

The causes of failure were suppuration of the cornea or whole eyeball, severe choroido-iritis, and such a condition of the deeper parts of the globe, that no improvement of vision resulted, though the cataract was removed without difficulty and no subsequent complication occurred.

My after-treatment of extraction cases is extremely simple; both eyes are bandaged as already described, and the lint is kept constantly wet by dripping cold, iced or warm water, upon the outside of the bandage as often as may be requisite. In most cases I use cold water, but if I have had any trouble during the operation, or for any reason expect inflammatory changes, a lump of ice is kept in the vessel from which the

water is taken ; in old and feeble persons I use warm water. At the end of the second day the pieces of lint are changed, but I never attempt to open either eye till the end of the first week, and not always then ; if the eyelids retain their natural appearance I am quite satisfied ; if the patient opens the eyes himself even so soon as the day after the operation I have no fear, for he will not do so unless all is going exceptionally well.

I keep the patient in bed for four days, at the end of the week let him have the sound eye untied, and wear a large green shade over both ; the eye operated upon is kept bandaged for three weeks, the lint being kept constantly wet for the first fortnight ; the shade is worn until all undue vascularity has subsided ; at the end of a month vision is carefully tested, and glasses are given about two months after the operation.

If, shortly after the operation, pain is complained of, and I notice that the eyelids are becoming red and swollen I suspect iritis or commencing suppurative corneitis ; I then order belladonna lotion to be used instead of water, apply leeches to the temples, and in some cases give mercury and opium internally. I also see that the lower lid is not inverted, but do not open the eye ; if entropion is found, strapping is applied so as to keep the lid in place ; this failing, a portion of skin and orbicularis muscle is removed.

Table of One Hundred and Fifty Operations for Extraction of Cataract.

No.	Sex.	Age.	Eye.	Form of cataract.	Operation.	Result.	Remarks.
1	M.	70	L.	Nuclear	Oblique corneal section downwards	Circular moveable pupil. S 2 C + 3½	Eye was myopic before operation; <i>no anæsthetic</i> .
2	F.	76	L.	"	Small flap downwards, with iridectomy	S 2 C + 2½, ⅔ C + 3	Had iritis for some weeks.
3	M.	70	L.	"	Small flap section upwards, iridectomy	S 1½ C + 2½, ⅔ C + 3½.	
4	F.	65	R.	"	Small flap downwards, preliminary iridectomy	S 2 C + 2½, ⅔ C + 3	
5	F.	"	L.	"	Ditto	S 3 C + 2½, ⅔ C + 3	Cataract immature in both eyes; preliminary iridectomy downwards in both eyes three weeks before extraction in first eye; <i>some vitreous lost in left; no anæsthetic</i> .
6	M.	73	R.	"	Small flap upwards; iridectomy	S 1½ C + 2, ⅔ C + 2½	High degree of hypermetropia; had worn × 9 for going about for some years.
7	F.	78	R.	"	Ditto	S 2 C + 3, ⅔ C + 4	Eye had been blind twenty years.
8	F.	68	R.	"	Linear section upwards, iridectomy	S 2½ C + 3, ⅔ C + 4	
9	M.	63	R.	"	Oblique corneal section downwards; no iridectomy	S 6 C + 2½	Had a violent attack of iritis, which left numerous adhesions of iris to parts behind; pupil opened with sharp hook; <i>no anæsthetic</i> .
10	M.	69	L.	"	Linear section upwards; iridectomy	S 4½ C + 2½, ⅔ C + 3. Counts fingers, and can tell one from the other	Severe iritis l.r.; pupil became entirely occluded; iris cut across from side to side with Wecker's scissors; clear pupil obtained.
11	M.	"	R.	"	Ditto		Preliminary iridectomy downwards in right eye at same time.
12	M.	57	L.	"	Small flap section upwards; iridectomy	S 2½ C + 2½, ⅔ C + 3	Vitreous bulged, but none escaped; iritis; pupil closed; iridectomy upwards.
13	M.	"	R.	"	Small flap downwards, iridectomy	S 6½ C + 2½, ⅔ C + 3	Vision was tested a week after operation in second eye, a fortnight after that in first. Patient died three weeks later, and post-mortem aortic aneurism was found. He took the anæsthetic very badly on each occasion.
14	M.	60	L.	"	Oblique corneal section downwards	Time on watch. C + 2½. Pupil circular	
15	M.	"	R.	"	Ditto		

No.	Sex.	Age.	Eye.	Form of cataract.	Operation.	Result.	Remarks.
16	M.	53	L.	Nuclear	Linear section upwards; iridectomy	S 2 C + 2½, ⅔ C + 3	Neither lens entirely opaque, but had no useful vision; iridectomy performed downwards in right eye at same time.
17	M.	"	R.	"	Small flap, section downwards; iridectomy had been already performed	S 2 + 2½, ⅔ C + 3	
18	M.	80	L.	"	Oblique corneal section downwards	⅔ C + 3	Sphincter of pupil removed, as it would not dilate to allow escape of lens; <i>no anæsthetic</i> .
19	M.	69	R.	"	Linear section upwards; iridectomy	S 2 C + 2½, ⅔ C + 3½.	Cataract immature in both eyes; no useful vision; iridectomy performed downwards in right eye at time of extraction in left. Pupil in left became blocked by inflammatory material, which was subsequently torn through with two needles.
20	F.	70	R.	"	Ditto	S 3 C + 2½, ⅔ C + 3½.	
21	M.	75	L.	"	Ditto	Can count and distinguish fingers; sees to go about	
22	F.	73	L.	"	Ditto	Both eyes can count and distinguish fingers; sees to go about	Both eyes were operated on at the same time; had entropion of both lower eyelids and severe iritis, which closed the pupils in both; operated on for entropion; subsequently double iridectomy in both eyes.
23	F.	"	R.	"	Oblique corneal section downwards		
24	M.	38	R.	Cortical	Small flap section downwards. Preliminary iridectomy ten months previously	S 1½ C + 4½, ⅔ C + 10	Both eyes stellate; opacity near anterior and posterior surfaces of both lenses; iridectomy downwards in both; improved vision for a time; always myopic; extensive atrophy of choroid in left. The right lens became rapidly entirely opaque ten months after iridectomy.
25	M.	14	R.	Cortical (lamella)	Small linear section upwards; iridectomy	Has never learnt to read; can see letters of 40 at 20 ft. C + 3	Patient has the rocky teeth described by Mr. Hutchinson. Had fits in infancy, for which he took powders. The left eye is affected, but has very fair vision.

NO.	SEX.	AGE.	LE.	CURABLE	OPERATION	REMARKS	RESULT
27	F.	30	L.	"	Small linear section upwards; iridectomy	Can count and distinguish fingers, and see to go about	from the operation the vitreous was found full of opaque floating clouds, probably hemorrhages; the cataract was probably secondary to choroidal disease.
28	M.	65	R.	Nuclear	Oblique corneal section	Never learnt to read; spells 50 at 20 feet C+3	The cataract was partially fluid, but there was a considerable gelatinous nucleus. Much opacity in vitreous was subsequently found; vision was improving when patient was last seen.
29	F.	63	L.	"	Linear section upwards; iridectomy	Only knows letters; can spell S 6½ C+4½, 28 C+5	Opaque capsule had to be torn through with two needles.
30	F.	48	L.	"	Small flap section upwards; iridectomy	S 3 C+4, 28 C+6	Some corneal opacity. Right eye myopic; much floating opacity in vitreous; large posterior staphyloma; very imperfect vision; iridectomy performed downwards at time of extraction in left.
31	F.	64	L.	"	Linear section upwards; iridectomy	S 1½ C+2½, 28 C+3	Patient always near-sighted; considerable corneal opacity in right eye.
32	M.	76	L.	"	Small flap section downwards; preliminary iridectomy	S 5½ C+2½	Two needle operations for opaque capsule.
33	M.	62	R.	"	Linear section upwards; iridectomy	Counts fingers; sees to go about	The pupil was entirely blocked and adherent to lens capsule. The right eye had been lost after extraction some years previously; globe shrunken to small irregular mass. Double iridectomy, upwards and downwards, in left; cataract extracted a month later; had iritis, followed by blocking of pupil; remains of iris removed; subsequently opaque capsule torn through with two needles; clear pupil obtained.
34 } 35 }	M. M.	60 ..	R. L.	" "	Ditto Small flap upwards; iridectomy	S 3½ C+2½, 28 C+3. S 2 C+2½, 28 C+3.	Closed pupil from iris after extraction; double iridectomy performed.
36	M.	58	L.	"	Oblique corneal section downwards. Sphincter of pupil removed	Cannot read; makes out the letters of S 1½ C+2½; spells 40 at 20 feet C+3.	<i>No anæsthetic.</i>

No.	Sex.	Age.	Eye.	Form of cataract.	Operation.	Result.	Remarks.
37	F.	66	L.	Cortical	Small flap section downwards; iridectomy	Cannot read; threads small needle C + 2½; spells 40 at 20 feet C + 3.	Patient was in an advanced stage of diabetes. Cornea suppurated. No anæsthetic.
38	M.	—	—	"	Ditto	Eye lost	
39	M.	50	R.	"	Oblique corneal section downwards. Sphincter of pupil removed	S 3 C + 2½, ⅔ C + 3	
40 } 41 }	M. M.	61 "	R. L.	" "	Ditto Small flap section downwards; iridectomy	S 1½ C + 2½, ⅔ C + 3½. ⅔ C + 4½.	Cataract shrunken, adherent; had to be removed by sharp hook; vitreous fluid, some escaped. Iridectomy had been performed some months before; cataract was secondary to choroidal disease. This patient had a severe attack of iritis in both eyes four months after the extraction of the cataract; the pupil was closed; iridectomy, and afterwards a needle operation, were performed. She can now see to go about, but cannot read.
42	M.	43	R.	"	Small flap section upwards; iridectomy	S 2 C + 2, ⅔ C + 2½.	
43 } 44 }	F. F.	60 26	R. L.	" "	Ditto Small flap section downwards; iridectomy	S 1½ C + 2½, ⅔ C + 3.	
45	M.	44	L.	Nuclear	Small flap section downwards; iridectomy	Bare perception of light	
46	F.	55	R.	"	Linear section upwards; iridectomy	Counts fingers; can find his way about S 3½, C + 2½, ⅔ C + 3	
47	F.	69	L.	"	Small flap upwards; iridectomy	S 1½ C + 2½, ⅔ C + 3	Had marks of past iritis in both eyes; lenses only partially opaque; no useful vision. Pupil became blocked by iritis after extraction; iridectomy upwards.
48	M.	31	L.	Cortical (traumatic)	Small linear section upwards; iridectomy	S 1½ C + 2½, ⅔ C + 3.	
49 } 50 }	F. M.	14 60	R. L.	" Nuclear	Ditto Linear section downwards; preliminary iridectomy	S 2½ C + 2½. Counts fingers; sees to go about	

51	M.	62	R.	Small flap section down-wards; iridectomy	S 3½ c + 4	Two needle operations for opaque capsule.
52	M.	64	R.	Small flap section down-wards; preliminary iridectomy	Counts fingers; sees to go about	Patient gouty; subject to iritis; pupil became blocked in right eye; iridectomy performed upwards; opaque membrane cut across with scissors; <i>no anæsthetic</i> .
53	M.	"	L.	Ditto	Eye lost	Left eye cornea suppurated; <i>no anæsthetic</i>
54	M.	73	R.	Small flap section up-wards; iridectomy	Ditto	Cornea suppurated.
55	M.	15	L.	Small linear section up-wards; iridectomy	S 2 c + 2½, ⅔ c + 3	Injury twelve months before operation, but cataract had only been noticed four months before.
56	F.	64	L.	Small flap section up-wards; iridectomy	S 2½ c + 2½, ⅔ c + 3.	
57	F.	62	L.	Ditto	S 3½ c + 2½, ⅔ c + 3.	
58	M.	19	R.	Small linear section up-wards; iridectomy	S 2 c + 2½, ⅔ c + 4.	
59	M.	57	R.	Large flap section down-wards; iridectomy	Distinguishes large objects, as hand, held in front of eye	Microphthalmos; coloboma of iris. Right eye blind forty years; left failed during last two years. Corneal opacity; small piece of iris removed by side of coloboma in right when extraction was performed; had iritis; pupil became blocked; iridectomy performed upwards.
60	M.	"	L.	Large flap section up-wards; iridectomy	S 12 c + 2½; sees to go about easily. c + 3	Left hemorrhage in vitreous some months after extraction. Coloboma of choroid in both eyes.
61	F.	3	R.	Small linear section up-wards; iridectomy	Pupil clear; optic disc visible; appears to see well; too young to be tested with lenses	
62	M.	57	L.	Small flap section up-wards; iridectomy	S 2½ c + 3, ⅔ c + 3½	
63	M.	5	R.	Ditto	Clear pupil; optic disc visible	Signs of old iritis.
64	M.	18	R.	Small linear section up-wards; iridectomy	S 1½ c + 2½, ⅔ c + 3.	
65	F.	58	R.	Oblique corneal section downwards	S 2 c + 2½, ⅔ c + 3.	<i>No anæsthetic.</i>

No.	Sex.	Age.	Eye.	Form of cataract.	Operation.	Result.	Result.
66	F.	26	R.	Cortical	Linear section upwards; iridectomy	Counts fingers; sees to go about	Iridectomy downwards; two needle operations on opaque capsule.
67	F.	57	R.	Nuclear	Small flap section downwards; preliminary iridectomy	S 12 c + 2½	Numerous posterior synechiae; pupil became closed after extraction; iridectomy upwards; needle operation on opaque capsule.
68	F.	62	R.	"	Ditto	½ c + 3.	
69	M.	67	R.	"	Small flap section upwards; iridectomy	S 1½ c + 2½, ½ c + 3.	
70	F.	52	L.	"	Oblique corneal section downwards; iridectomy	S 3½ c + 2½.	
71	F.	61	R.	"	Oblique corneal section downwards; preliminary iridectomy	½ c + 3½.	
72	F.	"	L.	"	Small flap section downwards; preliminary iridectomy	S 2 c + 2½, ½ c + 3	No anæsthetic. Vitreous fluid, some lost.
73	F.	72	R.	"	Ditto	Counts fingers; sees to go about	Left lens was removed with scoop, no vitreous lost; iritis; closed pupil; subsequent iridectomy upwards. Both eyes were myopic, and there were considerable atrophic changes in the choroid in left.
74	M.	60	R.	Nuclear	Linear section downwards; preliminary iridectomy	Counts fingers; can find his way about	Had severe iritis on several occasions before extraction. Both eyes were twice iridectomized; cataract was removed with sharp hook; subsequent severe attack of iritis; no anæsthetic.
75	F.	61	L.	"	Oblique corneal section downwards; small iridectomy	Counts fingers; sees to go about	
76	F.	66	R.	"	Small flap section upwards; iridectomy	S 1½ c + 2½, ½ c + 3.	
77	M.	67	R.	"	Small flap downwards; iridectomy	S 2 c + 2½, ½ c + 3.	
78	M.	19	R.	Cortical (traumatic?)	Iridectomy	S 2½ c + 2½, ½ c + 3½.	

79	M.	57	L.	Nuclear	Small flap section downwards; iridectomy	Counts fingers	Had severe iritis; subsequent iridectomy upwards. Both eyes had clear pupils, but patient was never tested with glasses, as he died from bronchitis three weeks after the last operation. Cornea suppurated.
80	M.	"	R.	"	Ditto	Ditto	
81	F.	75	R.	"	Small flap section upwards; iridectomy	Eye lost	
82	F.	"	L.	"	Ditto	Ditto	Choroido iritis.
83	M.	44	R.	"	Oblique corneal section downwards; no iridectomy	Cannot read; spells 30 at 20 feet C+3	No anæsthetic.
84	F.	63	R.	"	Ditto	S 2 C+3 $\frac{1}{2}$, $\frac{2}{10}$ C+5	No anæsthetic. Pupil circular, moveable. Had iritis; pupil became blocked; iridectomy downwards.
85	F.	60	R.	"	Small flap section upwards; iridectomy	Counts fingers; sees to go about	
86	M.	55	R.	"	Small flap section downwards; iridectomy	S 1 $\frac{1}{2}$, C+2 $\frac{1}{2}$, $\frac{2}{10}$ C+3.	
87	F.	48	L.	"	Small flap section upwards; preliminary iridectomy	Counts fingers; sees to go about	Had severe iritis; preliminary iridectomy was performed in both eyes.
88	M.	36	R.	Cortical	Small flap section upwards; iridectomy	Cannot read; tells time on watch C+2 $\frac{1}{2}$.	
89	M.	59	L.	Nuclear	Oblique corneal section downwards; preliminary iridectomy	S 2 $\frac{1}{2}$ C+2 $\frac{1}{2}$, $\frac{2}{10}$ C+4	Needle operation for opaque capsule.
90	M.	"	R.	"	Small flap section downwards; preliminary iridectomy	$\frac{2}{10}$ C+4.	
91	F.	80	R.	"	Small flap section downwards; iridectomy	S 2 $\frac{1}{2}$ C+2 $\frac{1}{2}$, $\frac{2}{10}$ C+3	No anæsthetic.
92	M.	70	R.	"	Ditto	S 1 $\frac{1}{2}$ C+2 $\frac{1}{2}$, $\frac{2}{10}$ C+3	No anæsthetic. Suppuration of cornea; no anæsthetic.
93	M.	"	R.	"	Ditto	Eye lost	
94	M.	50	R.	"	Small flap section upwards; iridectomy	$\frac{2}{10}$ C+3.	
95	F.	65	R.	"	Oblique corneal section downwards. Small iridectomy	Counts fingers; sees to go about	Pupil is partially closed; has granular ophthalmia, for which she is still under treatment; will probably gain excellent vision after iridectomy upwards.
96	M.	43	L.	"		S 3 C+3 $\frac{1}{2}$, $\frac{2}{10}$ C+6	
							Patient was myopic before extraction of cataract.

No.	Sex.	Age.	Eye.	Form of cataract.	Operation.	Result.	Remarks.
97	F.	28	L.	Cortical	Small flap section upwards; iridectomy	S 1½ c + 2½, ⅔ c + 3.	
98	M.	63	L.	Nuclear	Small flap section downwards; iridectomy	S 2½ c + 2½, ⅔ c + 3.	
99	M.	59	R.	"	Oblique corneal section downwards. Small iridectomy	S 2 c + 2½, ⅔ c + 3	No <i>anæsthetic</i> . Patient extremely unsteady; cataract removed with scoop; <i>some vitreous lost</i> .
100	M.	"	L.	"	Small flap section upwards; iridectomy	S 2½ c + 2½, ⅔ c + 3	Anæsthetic given; no difficulty.
101	M.	59	R.	"	Ditto	S 2½ c + 2½, ⅔ c + 3½	Lens shrunken; removed with scoop; no vitreous lost.
102	M.	36	L.	Cortical (traumatic)	Small flap section upwards	S 6½ c + 2½, ⅔ c + 3.	
103	M.	21	R.	Cortical	Small flap section upwards; iridectomy	S 6½ c + 2½, ⅔ c + 3	Cataract probably secondary to choroidal disease; some floating opacity in vitreous found after extraction.
104	F.	20	L.	"	Ditto	Entirely successful	Patient had diabetes, and left the hospital before being tested with glasses; clear pupils in both eyes, and she could see to go about.
105	F.	"	R.	"	Ditto	Ditto	No <i>anæsthetic</i> .
106	M.	53	R.	Nuclear	Small flap section downwards; iridectomy	S 2 c + 2½, ⅔ c + 3	
107	M.	"	L.	"	Ditto	S 2 c + 2½, ⅔ c + 3	
108	F.	28	L.	Cortical	Oblique corneal section downwards; preliminary iridectomy	Counts fingers; sees to go about; time on watch c + 2½	Ditto.
109	M.	60	R.	Nuclear	Small flap section downwards; iridectomy	S 1½ c + 2½, ⅔ c + 3	Cataract secondary to choroidal disease; right eye blind and shrunken; excised at same time as preliminary iridectomy in left; patient deficient in intellect; has never learnt to read.
110	M.	63	R.	"	Small flap section upwards; iridectomy	S 1½ c + 3½, ⅔ c + 4½.	Needle operation for opaque capsule; some floating opacity in vitreous.
111	F.	46	R.	Cortical (congenital)	Ditto	S 5 c + 16 D, ⅔ c + 13 D.	
112	F.	45	R.	Nuclear	Ditto	S 6 c + 13 D, ⅔ c + 12 D.	

1113 }	F.	28	L.	Cortical	Ditto	Counts fingers	} Both eyes were highly myopic, with extensive choroidal disease. Needle operation for opaque capsule. Ditto.
1114 }	F.	"	R.	"	Ditto	Ditto	
1115 }	M.	57	R.	Nuclear	Small flap section down-wards; iridectomy	S 1 c + 16 D, S 6 c + 13 D	
1116	F.	61	L.	"	Ditto	Sees figures on watch; cannot tell time	Right eye myopic.
1117	F.	50	R.	"	Small flap section up-wards; iridectomy	No useful vision	Severe iritis; iridectomy upwards; pupil again became closed; double iridectomy inwards and outwards.
1118	M.	40	L.	"	Ditto	S 5 c + 12 D, S 6 c + 10 D.	Much vomiting during operation, and, subsequently, vitreous bulged, but none escaped. Cicatrix bulging; pupil drawn towards section and blocked; iridectomy downwards; pupil again closed.
1119	F.	63	L.	"	Ditto	S 15 c + 16 D.	
1120	F.	42	L.	"	Ditto	No useful vision	
1121	M.	51	R.	"	Oblique corneal section downwards; small iridectomy	S 6 c + 16 D, S 6 c + 13 D	Much straining during operation; small quantity of vitreous lost. <i>No anæsthetic.</i> Needle operation for opaque capsule.
1122 }	M.	60	L.	"	Small flap section up-wards; iridectomy	Cannot read, spells 18 at 6 metr.; c + 13 D.	Eye became glaucomatous; large iridectomy upwards; relief of tension.
1123 }	M.	"	R.	"	Small flap section down-wards; iridectomy	Counts fingers	
1124	M.	40	L.	"	Ditto	S 5 c + 16 D, S 6 c + 13 D.	
1125	F.	55	R.	"	Ditto	S 6 c + 16 D, S 6 c + 13.	Severe iritis; pupil closed; iridectomy downwards; pupil again closed; double iridectomy inwards and outwards; opaque membrane torn through with sharp hook; clear pupil.
1126	F.	70	R.	"	Small flap section up-wards; iridectomy	S 4 c + 16 D.	
1127	F.	60	L.	"	Ditto	S 6 c + 16 D, S 6 c + 13 D.	
1128	M.	18	L.	Cortical (traumatic)	Ditto	S 5 c + 12 D, S 6 c + 11 D.	

No.	Sex.	Age.	Eye.	Form of cataract.	Operation.	Result.	Remarks.
129	F.	67	L.	Nuclear	Small flap section downwards; iridectomy	S 1 c + 16 D, 1/2 c + 13 D	No anæsthetic.
130	F.	64	L.	"	Ditto	S 5 c + 16 D, 1/2 c + 13 D	Ditto.
131	M.	60	R.	Nuclear	Small flap section upwards; iridectomy	S 5 c + 16 D, 1/2 c + 11 D	
132	M.	61	L.	"	Ditto	S 5 c + 16 D, 1/2 c + 13 D	
133	F.	64	R.	"	Ditto	Cannot read: counts S 12 at 6 meters c + 13 D; time on watch c + 16 D.	
134	F.	63	R.	"	Ditto	S 5 c + 16 D, 1/2 c + 13 D	
135	M.	61	R.	"	Ditto	S 5 c + 18 D, 1/2 c + 10 D	
136	M.	24	L.	Cortical	Ditto	S 5 c + 16 D, 1/2 c + 13 D	
137	M.	49	L.	Nuclear	Small flap section downwards; iridectomy	Can find his way about; cannot count fingers	Pupil closed by iritis; iridectomy upwards. Right eye had been excised some years before; probably choroidal diseases in left; much opacity in vitreous.
138	F.	94	R.	"	Small flap section downwards; preliminary iridectomy	S 5 c + 13, 1/2 c + 11.	
139	F.	58	L.	"	Ditto	S 5 c + 16 D, 1/2 c + 13 D.	Two needle operations for opaque capsule.
140	M.	55	R.	"	Small flap section upwards; iridectomy	Eye lost	Suppuration of cornea.
141	F.	66	R.	"	Ditto	S 5 c + 16 D, 1/2 c + 12 D.	
142	F.	60	L.	"	Ditto	S 5 c + 16 D, 1/2 c + 14 D.	
143	F.	58	L.	"	Ditto	Time on watch: c + 8 D, 1/2 c + 3 D.	Highly myopic.

144	M.	40	L.	"	Ditto	S $\frac{1}{8}$ c + 16 D, $\frac{1}{8}$ c + 13 D.	<i>No anæsthetic.</i>
145	F.	68	L.	"	Ditto	S $\frac{2}{8}$ c + 16, $\frac{1}{8}$ c + 13 D.	
146	M.	68	R.	"	Small flap section down- wards; iridectomy	S $\frac{1}{8}$ c + 15 D, $\frac{1}{8}$ c + 12 D.	
147	M.	78	L.	"	Small flap section up- wards; iridectomy	S $\frac{1}{8}$ c + 16 D.	Lens became displaced behind; iris removed with scoop; no vitreous lost.
148	F.	60	L.	"	Ditto	Time on watch, c + 16 D.	
149	F.	68	L.	"	Ditto	S $\frac{2}{8}$ c + 16 D, $\frac{1}{8}$ c + 13 D.	
150	M.	68	R.	"	Small flap section down- wards; iridectomy	D $\frac{1}{8}$ c + 13 D, $\frac{1}{8}$ c + 12 D.	<i>No anæsthetic.</i>

A CASE
OF
SECONDARY TREPHINING
FOR
TRAUMATIC ABSCESS OF THE BRAIN.
RECOVERY.

BY
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(Received February 11th—Read March 11th, 1879.)

THE bearing of this case on the diagnosis of traumatic abscess in the brain, and on secondary trephining, has appeared to me to impart to it sufficient interest to justify me in bringing it under the notice of the Royal Medical and Chirurgical Society.

An errand boy, æt. 15, hurrying out of a factory at the ringing of the dinner-bell, pushed by a shop-mate, stumbled against an iron fence, by which his forehead was grazed nearly in the middle line, a little below the hair.

Stunned for a few moments he soon regained his senses, and walked to the Middlesex Hospital to have the graze dressed. It was deep, but it did not lay bare the bone.

After this he went back to the factory and stayed there till evening, when, as he still felt shaken by the accident, a companion took him home.

He continued at his employment during the next month, but throughout this time he had more or less constant pain in the forehead, and he sometimes felt so ill that his master would dismiss him early in the day before the factory closed.

The graze festered, and it healed slowly. A month after the date of the injury he began to be troubled with nausea, and soon after to occasionally vomit without, he said, having done anything to provoke this.

One week later (five weeks after the injury), on 27th September, 1875, he was admitted into Percy Ward, Middlesex Hospital, under the care of Mr. Andrew Clark, who had charge of my beds during my vacation. His symptoms being considered indicative of intracranial inflammation leeches were put on the forehead and his head was kept cool with ice.

On October 10th, when he came under my care, he was lying on his back, his eyes shut; he seldom, I was told, moved, and he took little notice of what was occurring in the ward around him. When questioned he always complained of pain in the forehead, where the situation of the graze was marked by a small scar, tender, and tied to the underlying bone. His tongue was furred, slightly brown, and dry. He disliked eating, and he often retched. His skin felt dry and rough, but it was cool. Next day at noon I found him lying on his left side, and then first detected incomplete hemiplegia of this side. He could not grasp as strongly with the left hand as with the right, and the strength of his left thigh, roughly measured by holding it down on the bed whilst he tried to flex it, was plainly less than that of his right thigh.¹ His pulse was only 48 per minute, and the temperature in the armpit was 97·8° F.

Believing an abscess to have formed in the right frontal

¹ My colleague, the late C. De Morgan, confirmed the existence of the hemiplegia.

lobe of the brain, I decided to trephine at the scar of the graze and make an outlet for the pus.

Chloroform was given, for although somewhat heavy in manner the boy was perfectly conscious.

When the coverings, including periosteum, were turned back, preparatory to setting on the trephine, a small starred crack in the outer table of the frontal bone became apparent. It occasioned no unevenness of the outer surface, and it did not penetrate to the inner table. A disc of bone $\frac{7}{16}$ inch diameter was cut out. About $\frac{1}{3}$ (the left) of its inner surface was rough. This was evidently merely the mark of the more intimate adhesion of the dura mater bounding the superior longitudinal sinus which was exposed at the extreme left of the bore-hole. At the right of this the dura mater bulged up tensely into the bore-hole, but its outer surface appeared healthy, and there was not anything observable which might denote abscess beneath it in the subdural space or under the (visceral) arachnoid. The cerebral pulsation was apparent. In the expectation that the pus was more deeply placed, a fine trocar, connected with a partly exhausted aspirator, was now pushed through the membranes into the brain at the right of the bore-hole, and when it had reached a depth of about one inch, thin greenish pus rose into the syringe. The rough characters of the fluid were unmistakable, and they were afterwards confirmed by microscopic examination. A free opening into the abscess was next made with a narrow knife passed upon the trocar as along a guide, and through this opening, and also through the trocar, a quantity of pus, estimated at between ʒiij and ʒiv escaped. While enlarging the opening, although care was taken to keep the edge of the knife as far as possible from the situation of the longitudinal sinus, a minute slit was made in its extreme border from which a fine stream of blood spurted to a distance of about one foot. Slight pressure with the finger tip stopped its flow, but the paramount necessity of leaving the bore-hole quite free for the drainage of the abscess forbade the use of a compress. The lips of this little slit were, therefore, transfixed with a very

fine suture, in passing which care was taken to avoid including the lining, the stitch was knotted and then a thread was tied circularly round, the slit being kept from slipping by the knot which acted as a button. This little manœuvre is mentioned only as a hint to others who may meet with a similar difficulty. I had several years before found it efficient in the arrest of bleeding from a small vein divided close to its termination in the axillary trunk. During the operation and subsequently antiseptic precautions were strictly observed.

The immediate effects of the operation were the disappearance of the hemiplegia and the cessation of the retching. The pain in the forehead subsided slowly. During the first fortnight the pulse rate ranged between 48 and 55, and the temperature was generally below the normal average. There was a slight escape of pus from beneath the skin-flap which had been loosely replaced over the bore-hole, but this ceased after a few days, and before the end of October the wound had closed.

Notwithstanding this local progress the boy's general condition for a long time was unsatisfactory. Although his appetite returned, so that he ate with relish a mutton-chop for dinner, yet he lost flesh. He himself invariably told me that he felt better.

November 7th, nearly one month after the operation, the ward-sister noticed that he seemed to grope upon his plate for his food as if he did not plainly see it. I now found that the visual acuteness of both eyes was blunted and detected in both the characteristic signs of neuro-retinitis. For this mercury was rubbed in until the gums were slightly touched, and its influence was gently maintained for one month. Under this treatment the sight of the right eye improved so much that he became able with it to read small print and see the hands of a watch at the distance of one foot; but the improvement did not continue, and eventually both optic papillæ underwent atrophy, and both eyes became blind.

At the end of December he left the hospital in fair general health. About six months later he was brought again on

account of fits. They were probably epileptic. Potassic bromide was prescribed with relief. I saw him last summer, he had become stout and looked hearty. He had not had a fit for a long time. He was then under instruction in an industrial school, and I was told that he was an intelligent, well-conducted pupil.

In injuries of the skull affecting its contents, greatly as surgeons have differed respecting other indications for trephining, all, I believe, have ever agreed that it is proper to trephine where this is necessary to afford escape to the contents of an abscess in the brain. In the application of this precept uncertainty of diagnosis constitutes a real difficulty which has injuriously limited its practice. The question whether an intra-cranial abscess can be inferred with so high a degree of probability as to justify so grave a proceeding as trephining has been differently answered by surgeons of great experience.

Not to refer to writings of past times, in our own day we find one eminent authority formulating groups of symptoms by the aid of which not merely may suppuration inside the skull be recognised, but the diffused distinguished from the localised, and the depth of the abscess gauged, whether in diploë, between inner table and dura mater, in subdural space, under the (visceral) arachnoid, or, lastly, in brain. Another equally eminent authority diffidently acknowledges his inability to lay down clinical characters by which the diffuse can be discriminated from localised suppurations within the skull, and the seat of the latter inferred with more than relatively slight probability.

In the case just narrated hemiplegia was the symptom which decided me to trephine in the belief of the presence of abscess in brain. I am aware that a very distinguished clinician has expressed the opinion that hemiplegia (when separated by so great an interval of time from the date of injury that it cannot be properly regarded as the immediate and direct result of brain-lesion) is symptomatic of arachnitis; but I submit that when hemiplegia supervenes after such an interval of time it is significant of a morbid process

proceeding in the brain itself rather than in its membranes, and when inflammation of these occurs it is secondary and of subordinate importance. The following case, which I had an opportunity of watching when attached to the General Hospital in the front before Sebastopol, early impressed me with this.

A private in a line regiment was struck by a glancing rifle-bullet on the vertex just to the right of the middle line. The scalp was scored, but the bone was thought to have escaped injury. The eschar fell, the wound granulated and had nearly healed, leaving, however, a small sinus. Throughout this time there were not any brain symptoms. He then complained of not feeling well, his indisposition increased; violent pain in the crown of the head set in; he began to retch; and next day his left arm was palsied. Bare bone being now felt on probing the sinus it was decided to trephine. When the scalp had been turned back the upper and posterior angle of the right parietal bone was found splintered and depressed. Some loose splinters were removed and the depressed bone raised. The exposed dura mater had not been torn, and it did not exhibit any indication of disease. Nothing further was done. The symptoms continued unrelieved; delirium and great fever shortly followed and death took place on the second day after the trephining.

At the necropsy, on examining the head an abscess was found in the right cerebral hemisphere directly beneath the fracture, close to the surface of the brain. From this spot, as from a focus of infection, the web of the pia mater was infiltrated to a considerable distance with a greenish sero-purulent fluid. It was a matter of regret that, on not finding pus between the bone and dura mater, the surgeon had not cut through this into the brain; in the patient's desperate state it would have added little to his danger, and it might have afforded him a chance of surviving. The rather sudden and great accession of fever after the operation marked, it was thought, the onset of the purulent meningitis which might have been averted by the timely evacuation of the abscess.

CASES
OF
PERFORATING ULCER OF THE FOOT,
WITH REMARKS, AND AN APPENDIX ON THE
LITERATURE OF THE SUBJECT.

BY
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THE curious disease known under the somewhat misleading title of perforating ulcer of the foot has hitherto attracted comparatively little attention in this country, and is hardly alluded to in our surgical literature. On the Continent, and especially in France, where it is known by the name of "mal perforant du pied," it has been more generally recognised and described by many surgeons. But the majority of these essays deal only with certain of the more prominent features of the disease, and, with a few important exceptions, scarcely, if at all, touch the great fact in its pathology. At the end of this paper we have drawn up a list of the principal articles which have appeared on the subject, with a brief summary of the chief points in those which seem to us most worthy of study. We propose now

first to relate some cases in illustration of this disease; then to point out, so far as we are able to do so, its principal characters; and, finally, to discuss, by the light of the facts at present known to us, its pathology.

CASE 1.—George S—, æt. 40, was admitted on the 1st of September, 1877. Five years ago, when he was a ballet-dancer, a corn beneath the right great toe became sore and discharged. He then went into King's College Hospital. After poultices a piece of bone came away from the opening opposite the metatarso-phalangeal joint. He left the hospital with the wound closed and the joint stiff. As he could no longer dance he became a gilder.

Two years ago several corns that had formed on the feet began to be sore. These have steadily grown worse, so that it gives him much pain to attempt to walk. Lately he has noticed an eruption on the legs, and sweat often stands in large drops on them.

He is a dark, lean, ill-nourished man, very excitable. In the sole of each foot are three perforating ulcers. The disease is almost exactly symmetrical. One is placed in the middle line of the sole, and one on either side of this, corresponding respectively to the metatarso-phalangeal joint of the great and little toe. The same description applies to both feet. The right leg is somewhat wasted, and measures one inch less than the left round the calf. The extensor muscles of the foot are powerless; the foot falls forwards and inwards; he cannot extend the toes or flex the foot. Power over the left leg natural. A chronic eczema affects both legs, but more extensively the right one. Loss of sensation over both feet and legs; almost total insensibility of the skin of the dorsum of right foot. The legs and feet sweat continually. The arteries are soft, but the pulse in the right foot is very small, very much less than that in the left. Poultices.

October 5th.—The openings have closed, but the skin remains much thickened at the margin of the ulcers. He was discharged.

CASE 2.—Charles M—, æt. 44, a stableman, was in the hospital from November 21st, 1872, till January 26th, 1873, when the following note was made:—"Perforating ulcer of the right sole of some three years' duration, supposed to have originated in a suppurating corn. January 13th.—Healthy healing ulcer over the metatarsal bone of the fifth toe. Two or three pieces of necrosed bone were removed soon after admission. 26th.—Ulcer healed."

He was again admitted on September 14th, 1876. The scar of the wound is covered with horny epidermis like the surface of a corn. There is a similar hardened spot beneath the head of the fifth left metatarsal bone, in the centre of which is a small conical depression. On the under surface of the tip of the right second toe is a horny thickening from two to three lines across. Its centre is slightly excavated, and of a dusky-red colour, from the injected red capillaries beneath. A spot, exactly similar in appearance, though much larger, is seen on the under side of the right great toe opposite the phalangeal joint. These spots have appeared lately, and that on the right toe has discharged occasionally a thin fluid. On the corresponding part of the left great toe there is an ulcer about the size of a shilling, bounded by thickened epidermis, and having in its middle a small elevation formed by fungous granulations. The ulcer is of a year's duration, and was at first like that on the right great toe. A probe passed into the central elevation goes down to rough bone.

September 27th.—The left great toe was amputated by anterior and posterior flaps at the metatarso-phalangeal joint. The joint was denuded of cartilage and the bone carious.

From October 1st to October 9th an attack of erysipelas, which extended as high as the groin.

On October 31st the wound was healed, and he was discharged.

CASE 3.—Edwin P—, æt. 41, a porter, swarthy, muscular, temperate, and of good health, was admitted into the hospital

on July 24th, 1877. He says the sole of the right foot has been sore for nearly twelve years. At first he noticed a corn beneath the end of the fifth metatarsal bone. From time to time gatherings have formed and broken about that part of the foot. He was once in an infirmary for the disease, and whilst there the openings closed.

He has a perforating ulcer of the foot. The opening in the sole is opposite the head of the fifth metatarsal bone. It is large enough to admit a probe, and stands in the centre of thickened corn-like cuticle. The probe strikes bare bone. Over the dorsal surface of the foot the skin is reddened and glazed. The inner limit of this injected area is marked by the tendon of the extensor proprius pollicis; the posterior limit runs in front of the ankle and outer malleolus to the border of the foot.

A small scar on the dorsal surface opposite the opening on the sole of the foot marks the site of an old abscess. Over the injected area on the dorsum the sensibility is impaired, but not abolished. The foot sweats very little. The peripheral arteries are rather firmer than natural. There is no evidence of visceral disease in chest or abdomen. Urine 1030; no albumen; abundant phosphates.

On August 1st the little toe and metatarsal bone were amputated.

8th.—There has been considerable inflammation of the wound and dorsum of the foot since the operation. It extends over, but is confined to, the congested area mentioned above.

September 7th.—A small abscess on the inner side of the dorsum was opened.

14th.—The wound has nearly healed. There is still some swelling of the dorsum of the foot and the skin is dusky. He left the hospital.

CASE 4 (for which we are indebted to Mr. Smith).—Thos. B—, æt. 27, a clerk, admitted into the hospital May 19th, 1875, on account of a large and deep ulcer of the sole of the right foot. The ulcer was circular, about $1\frac{1}{2}$ inch

across, situated in the middle and inner portion of the sole, not far from the base of the toes. It was surrounded by a margin of thick, hard epidermis, resembling a corn; its surface was indolent and without granulations; there was no surrounding area of inflammation. Although the leg, ankle, and heel were well formed, the foot was strangely deformed. It was much shortened; the toes were small, stunted, and twisted; the front part of the foot was thickened. There were two small fistulous openings on the dorsal aspect. He complained of much pain in the ulcer, although examination of it gave him very little pain, and there was complete anæsthesia of the whole of the foot and lower part of the leg, and partial anæsthesia as high up as just above the knee. The movements of the ankle were perfect, and the patient was able to walk considerable distances. He was a healthy-looking man, with no other deformity. He said the right foot and leg had been "*numb*" from his earliest infancy, that his mother attributed this to a severe blow which she had received during pregnancy upon the right leg, and which had deprived her of feeling for a short time. The limb was, however, well-formed, and as good for all purposes of locomotion as the left. Fifteen years ago a heavy man, disbelieving his story of the numbness of his foot, put it to the test by treading upon the foot with his whole weight. Shortly after this occurrence an ulcer formed under the ball of the great toe. This gradually spread and affected the bones of the foot and toes, many of which came away piece by piece at irregular intervals. There was no family history of any similar affection. The leg was amputated a little below the knee.

The patient made a good recovery and has remained well up to the present date (January, 1879). The anæsthesia has not spread further up the limb.

Examination of the limb after removal showed no apparent alteration of arteries or nerves. The bones of the foot and toes were so much altered in shape and position that they could hardly be identified. The joints were destroyed, some of them ankylosed. The compact tissue of the tibia

was thickened and irregular. Most of the muscles and tendons of the foot were destroyed.

Microscopical examination of the ulcer showed the ordinary characters of a simple chronic ulcer. Transverse sections of the anterior and posterior tibial nerves as high up as possible exhibited great thickening of the epineurium, very little alteration of the perineurium, but thickening of the endoneurium in some of the fibres (Plate IV, figs. 5, 6, 7). In other fibres the endoneurium was normal, but the fibres were composed only of fibrils of the largest size (Plate IV, fig. 4) containing large axis-cylinders and a very thick coating of medullary substance. The fibres of both nerves were few in number and all of small size. There were very few *small* fibrils in any of the fibres. There was no increase of nuclei. The vessels appeared to be normal or but slightly thickened.

CASE 5.—James C—, æt. 27, an ostler, was admitted in November, 1875, on account of a suppurating corn, which he had first noticed in October, beneath the fifth metatarsal bone.

The corn was perforated in the centre, and from the hole a small quantity of sero-purulent fluid exuded. A probe passed into the substance of the fifth metatarsal bone, which was evidently carious. When asked if the foot had received any injury he said both his feet had often been trodden on by horses.

In the middle of December the fifth metatarsal bone was excised. The wound healed, and he left the hospital in January, but in February it broke out again, and he was readmitted early in March. He was kept in bed, and the ulcer on the sole was poulticed. It slowly, but at length completely, healed, and he left the hospital in the middle of April. The foot was then slightly inverted so that the weight of the body fell partly upon the outer edge.

At the beginning of April he first noticed excessive perspiration of the foot both by day and night. This gradually grew worse. When he resumed work the foot became weaker and more inverted.

On the 9th of June, 1876, he was seen again. The

perspiration of the foot was then very marked. Large drops stood on the outside and dorsum. And now there was partial but well-marked anæsthesia over the region of the two outer toes, and to a less extent over the whole dorsum of the foot. It was most marked over the metatarsal bones. There also the perspiration was most abundant. He complained of slight pain over almost the whole of the foot. There was a small hole (rather than an ulcer), with hardened skin around, on the outside of the sole, and a probe through this passed into the substance of carious cuboid.

The foot was amputated at the ankle-joint by Syme's operation. The patient left the hospital on the 11th of August.

On the 15th of September he was again admitted on account of an abscess along the course of the peroneal tendons following a fall upon the stump. Discharged October 4th well.

He remained well until Christmas, when a small abscess formed at the outer part of the stump, burst, and left an opening which would not heal. For this he was admitted into the hospital March 29th, 1877. There was a small ulcer, a quarter of an inch across, on the outer side of the face of the stump, circular in form, surrounded by thickened epidermis, and bearing all the characters of a perforating ulcer, but not exposing the bone. From the ulcer up the outer and back part of the leg as far as the junction of the middle and lower thirds of the thigh there was an area of anæsthesia, with very well-defined limits. A poultice was applied, and the ulcer healed, but when he left the hospital on the 28th of April there was a small abscess just above the ulcer.

On the 9th October, 1877, he returned to the hospital with the ulcer broken out again, begging to have the leg cut off. Amputation was performed through the tuber tibiæ in November, because the condition of the limb prevented him from working. He made a good recovery, and was discharged December 22nd.

The leg was carefully examined after removal. The ulcer

passed down between the tibia and fibula, but did not expose the bones. The main vessels and nerves appeared to be healthy, but the external saphena vein was completely obliterated by old disease. There was nothing abnormal in the condition of the bones or of the soft parts.

Microscopical examination was made of the ulcers, which bore the ordinary characters of simple chronic ulcers. Transverse sections were made of the anterior and posterior tibial nerves in the foot (removed by Syme's amputation), as far from the seat of disease as possible (Plate IV, figs. 8, 9, and Plate V, fig. 1). The connective tissue of all parts was much thickened, epineurium, perineurium, and endoneurium. The nerve fibres were smaller than is usually the case. The ultimate fibrils were generally atrophied, and large numbers of the smallest fibrils were completely obliterated (Plate IV, fig. 3), apparently by the increase of thickness of the endoneurium. There was not a very marked increase in the number of nuclei, nor were there any appearances leading us to believe that the nerve-sheaths were filled with nuclei. Examination of the same nerves in the upper part of the leg removed by the second amputation showed that they were there perfectly healthy in appearance, but the external saphenous and musculo-cutaneous nerves exhibited the same characters as those observed in the tibial nerves in the foot, although in a less degree. With the exception of thickening of the coats of the vessels in the tibial nerves in the foot the vessels in all the nerves appeared to be normal.

The disease as it appears in the foot is not accurately described by the term ulcer. It varies in character, and in some instances there may be granulations, occasionally even an exuberant mass of them, around the orifice; but usually a small aperture, like the orifice of a sinus, is seen in the centre of a large corn, and this leads directly down by a narrow channel to exposed and diseased bone. From this there is little or no discharge, and it is in all respects very indolent. There is no pain whatever when at rest; never

much even upon motion or pressure. Indeed, the ulcer itself is, as a rule, quite insensible. The aspect of the adjacent parts is quite unchanged, unless, as they are prone to be, attacked with inflammation; sometimes, however, the foot becomes livid when the temperature is reduced, and this is apt to occur, the surface being often very cold. There is usually anæsthesia, more or less complete, of this region, which terminates above, in most cases, perhaps, rather abruptly. All the portion of the limb thus affected, whether a part or the whole of the foot, with, perhaps, the lower part of the leg, is prone to sweat profusely, and the perspiration has sometimes a peculiarly fetid odour. These three features—loss of temperature, loss of sensibility, and tendency to profuse perspiration—are commonly well-marked, but there is usually no perceptible loss of motion.

The situation of the ulcer varies. Most frequently it is over the articulation of the metatarsal bone with the phalanx, oftenest over that of the first or last toes. It is rarely seen on the heel. There may be more than one; two or three on the same foot; or both feet may be affected, and then the disease may be symmetrical. It has also been seen on the hands.

The disease, even under unfavorable conditions, advances slowly, soon becomes apparently stationary under rest, and if this is prolonged the fistulous track may at length close, but only to break out again when the foot is brought into use.

With regard to the mode of formation of the so-called ulcer itself it would appear as if there were a gradual, but almost simultaneous, disintegration of tissue throughout the whole depth of the sole, from the skin to the bone. At all events there is no evidence to show that it begins at the surface and makes its way from without inwards, for when the aperture is discovered a probe will always enter to a considerable depth and will usually pass at once down to exposed bone.

Operations, whether for the removal of the diseased bone, or of a larger portion of the foot, are not, as a rule, permanently successful. The disease is apt to reappear at or

near the stump. It may be said that gradual, though obstinate progression, in spite of intervals of quietude or amendment, is one of its most significant characters. It occurs far more often in men than in women, sometimes in early life, and occasionally in the children of those who have suffered from it.

If called on to express an opinion of the nature of the disease, we should say that the so-called ulcer is the result of pressure, or violence, or injury to structures, whose nutrition is impaired or whose vitality is defective from disease or degeneration of the supplying nerves. In proof of this theory we would draw attention to the fact that marked changes have been discovered in the nerves passing to the affected part in every case in which they have been thoroughly examined; that the symptoms of nerve lesion in some cases have been noticed for a more or less considerable period before the appearance of the ulcer (as in the case of B—); and that an ulcer precisely similar in characters and course has been frequently described in connection with such diseases as locomotor ataxy, anæsthetic leprosy, and in limbs which have been long paralysed from disease of or pressure on the spinal cord. The nature and extent of these changes varies, as the description of the nerves in the cases of C— and B— shows. In the former (Plate V, fig 1) the endoneurium is much thickened and the degeneration or obliteration of nerve fibrils is most marked in the nerves near the ulcer, whilst the same nerves at a much higher point are free from disease. In the latter the nerves are abnormal as high up as they could be examined and the change in them consists not so much in a thickening of the endoneurium as in a complete absence of large numbers of fibrils (Plate IV, fig. 4) from fibres which otherwise appear perfectly normal in all their parts, a condition which forcibly suggests the idea of congenital deformity. The peculiar condition of the nerves, in this case especially, and the character of the symptoms of nerve lesion in most cases of perforating ulcer, lead us to attempt to explain, if possible, more exactly than has hitherto been done the relation which

may exist between these two states. The symptoms of nerve lesion in all cases point to affection of sensation and of nutrition, rarely to affection of motion; and never to affection of motion without affection of sensation or nutrition. In other words, the sensory and nutrient, or vaso-motor nerves, are at fault, the motor nerves generally unaffected.

It is of course impossible in the examination of a transverse section of a mixed nerve to decide whether any individual fibril is motor or sensory or nutrient.¹ But, inasmuch as the fibrils which compose the sympathetic nerve are for the most part small, and either non-medullated or almost without medullary sheath, it may be supposed that nutrient fibrils in mixed nerves bear similar characters, and that a certain number, therefore, of the smallest fibrils seen in a transverse section are nutrient.

There is, we are informed, an unpublished observation of Schweigger-Seidel, to the effect that sensory fibrils may be distinguished from motor fibrils by the thinner medullary sheath by which they are surrounded. We have tested this observation by an examination of sections of motor and sensory nerves taken from the same body; and, although it does not appear to be absolutely true, inasmuch as motor nerves contain thinly medullated fibrils, and sensory nerves contain some thickly-medullated fibrils, there is generally so distinct a difference that one can recognise with ease a motor or sensory nerve (Plate V, figs. 5—8). The fibrils of the former are larger, more thickly medullated, and more uniform in appearance. We believe, therefore, that the smallest fibrils of a mixed nerve are chiefly nutrient and sensory, and that the larger fibrils are chiefly motor.

The histological characters of the nerves in the two cases examined by us strengthen this belief. Both are characterised by absence of, or damage to, or obliteration of, the small fibrils, either from defect, which is perhaps congenital, or from disease, whilst the large fibrils remain appa-

¹ *I. e.* vaso-motor, or, perhaps, trophic.

rently intact. In the case of C—, this effect is produced by a uniform and general thickening of the endoneurium, a condition which, whenever it exists without a corresponding increase in the size of the nerve, must necessarily produce a similar effect. This thickening of the endoneurium and consequent degeneration of nerve fibrils is described in cases of perforating ulcer by Michaud, by Sonnenburg, and by Duplay and Morat, the last named, however, regarding the nerve degeneration as dependent, in the cases which they relate, upon other causes than the connective-tissue thickening. It is described also by Virchow¹ and by Langhans² in cases of anæsthetic leprosy as a constant lesion in that disease; and, as perforating ulcer occurs not uncommonly as a symptom of anæsthetic leprosy, this condition of nerve may be again regarded as the proximate or predisposing cause of the ulcer.

We should say, then, that the defects of sensation and nutrition which are observed in connection with perforating ulcer, and of which latter the ulcer is one of the most marked symptoms, are due to absence or degeneration of the sensory and nutrient fibrils of the supplying nerves; and that, in many cases of peripheral disease, at least, these fibrils suffer as the direct result of mechanical pressure produced by increase of the endoneurium of the nerves, whilst the motor fibrils escape owing to their larger size and thicker medullary sheath.

The causes which will produce this thickening of endoneurium are probably numerous. We have found it several times in the nerves of limbs which were the seat of rigid or calcareous vessels, and thus may be explained the relation which has been supposed by some authors to exist between perforating ulcer and arterial disease. In the case of C— we can find no apparent cause.

With regard to treatment it cannot be said that by any means at present known there is a fair prospect of permanent cure. In too many cases after operation, whether excision of

¹ Virchow, 'Krankhaften Geschwülste.'

² Langhans, Th., 'Virchow's Archiv,' 1875, Band 164, s. 169.

the soft structures affected, or of the diseased bone, or amputation of a toe, or of a portion of the foot, the disease has gradually returned in some neighbouring parts, so that the extreme remedy is at best a doubtful one. In one remarkable instance (C—'s) this affection obstinately recurred after repeated removal. By prolonged rest in the horizontal position many of these ulcers in the earlier stages will close, but only to break out again when the foot is used. In view of what is known of the pathology of this disease this obstinacy is not hard to understand. When previous excision has failed, or the disease has become established, it appears to us that the only alternative to the severe measure of amputation beyond the region affected with anæsthesia—and even that may fail—is the use of an artificial leg attached to the bent knee, so that the foot may be carried without having to take any part in supporting the weight of the body.

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Although descriptions which tally well with many of the descriptions of this disease occur in the works of such authors as Marjolin (1), and Boyer (2), and although writings still earlier than these speak of the

and sinuses which may occur in connection with corns.¹ M. Nélaton (3) first distinctly drew attention to perforating ulcer of the foot. In the case described by him the ulcers were said to commence beneath a bleb, and no mention is made of a corn. Both feet were affected, and the disease was hereditary. Almost immediately after this paper appeared, a paper by Vésignié (4), describing the disease in greater detail, and its commencement beneath a corn. He believed it originated in a sort of plantar psoriasis, and recommended the name "*mal plantaire perforant*," which has formed the basis of the nomenclature since in use. The thesis of Leplat (5) contained a very good account of the origin and course of the ulcers which he thought due to pressure effects, but that a special predisposition was necessary. M. Demarquay (13) first drew attention to the anæsthesia which so often accompanies perforating ulcer, and at a later date (17) published a case in which the urine had been examined for sugar, with a negative result, in order to see whether there was a relation between perforating ulcer and diabetic gangrene.

In 1863 M. Péan (18) brought before the Société de Chirurgie a case in which he had examined the affected limbs and had found extensive arterial disease—calcareous, and partially or wholly obstructed vessels,—and he naturally enough attributed the ulcers to this cause. This opinion was supported by Delsol in 1864 (24), and later by Dolbeau (27) and his pupil Lucain (31), both of whom employed the sphygmograph to prove arterial disease; the latter also bringing forward six cases, in which disease of the vessels had been verified by actual observation. But this theory was soon shaken by the relation of cases by Sédillot (23), Guyon (28), and others, in which the vessels were normal, and since that time it has not received any important support.

The occurrence of ulcers in leprous patients precisely similar to the perforating ulcer, was mentioned in 1864 by M. Poncet (19), and the description and sketch which were given in his paper made it certain that the

¹ Cloquet (0).

leprous ulcer was really a perforating ulcer. But it was not till much later (1871) that an attempt was made by Estlander (34) to prove that all perforating ulcers were due to leprosy, and were some of the last remnants of that disease in countries where it had formerly existed.

In spite of the strong opinion, expressed by many surgeons, that the disease was peculiar, and was due to some peculiar and uncommon cause, Larrey (12), Follin (22), Sédillot (23), Gosselin (26), and Pitha (32), still maintained that nothing more was necessary for its production than pressure and inflammation.

But in 1872 Poncet (37), again referring to the relation which exists between leprosy and perforating ulcer, described the condition of the nerves in a case of perforating ulcer, stating that the connective tissue was thickened, the nuclei too numerous, and the fibrils atrophied.

During the course of the next year appeared an admirable paper by Duplay and Morat (40), drawing attention especially to the symptoms of nerve lesion which accompany perforating ulcer, describing the microscopical characters of all parts affected by the disease, and more particularly the condition of the nerves, the fibrils of which were found in six cases exceedingly degenerated as far up as they could be examined.¹ The conclusions to which they were led were, that the perforating ulcer is due to a degenerative lesion of the nerves of the region; that this degenerative lesion may be due to diverse causes, lesion of the cord or spinal ganglia, section and compression of large nervous trunks, alterations of the nervous extremities, &c.

This neuro-pathology has steadily gained ground since 1873. Cases have been published, in which the nerves have been examined and found to be diseased, by Mazzoni (41), Fischer (44), Paul Bruns (43), Michaud (49), and Sonnenburg (51), all of whom are agreed in believing that the ulcer is due to nerve lesion, although there are differences

¹ The degenerative lesion was in every way comparable to that which is produced after section of nerves and their separation from the trophic centres.

of opinion as to the nature of the lesion with which it is associated. Michaud, in particular, differs from Duplay and Morat in the description he gives of the nerve lesion and the deductions he draws from the appearances observed. He found sclerosis of the nerves near the seat of disease, but the same nerves at a much higher point were normal. He therefore divides the ulcers into two categories. 1. Those which are symptomatic of various affections of the spinal cord or nerve trunks. 2. Idiopathic perforating ulcer, the true perforating ulcer, such as is described by Nélaton. "After the autopsy, of which we have given the details," he proceeds, "it does not appear to be due to a degenerative neuritis; one only finds a sort of peripheral sclerosis of nervous ramifications." Michaud and Paul Bruns are the only authors who mention the immunity from motor paralysis, which is so marked a feature in most cases, especially in those of peripheral origin. The theory of Schoemaker (42), that perforating ulcer is in most cases an epithelioma, is not borne out by facts.

We have endeavoured in the present paper to show that the disease is always symptomatic of nerve lesion, central or peripheral, and to explain why certain symptoms follow certain nerve-lesions.



DESCRIPTION OF PLATES IV, V, AND VI.

(Perforating Ulcer of the Foot. Mr. Savory and Mr. Butlin.)

PLATE IV.

Figs. 1—4. Transverse sections of nerve fibres $\times 85$. (Oc. 3, obj. 4, tube drawn out.)

Fig. 1. Post tibial, from patient with very calcareous vessels.

2. „ normal.

3. „ (C—), perforating ulcer.

4. „ (B—) „ „

Figs. 5—7. Posterial tibial nerve (B—), perforating ulcer.

Fig. 5. Section of nerve, very low power (A. 4 in.), large quantity of connective tissue, few fibres and small.

6. Portion of section highly magnified. $\times 260$.

7. Another portion $\times 260$.

Figs. 8, 9. Transverse sections of nerves (C—), perforating ulcer.

Fig. 8. Anterior tibial nerve.

9. Posterior „ very low power (B. 4 in.).

PLATE V.

Fig. 1. Transverse section of nerves (C—), perforating ulcer. Portion of post-tibial nerve $\times 260$, showing thickening of connective tissue and destruction of nerve fibres. (Oc. 3, obj. 7, tube drawn out.)

Figs. 2—4. Sections of normal nerves.

Fig. 2. Post-tibial nerve.

3. Ante-tibial nerve with very low power (A. 4 in.)

4. Portion of post-tibial nerve $\times 260$.

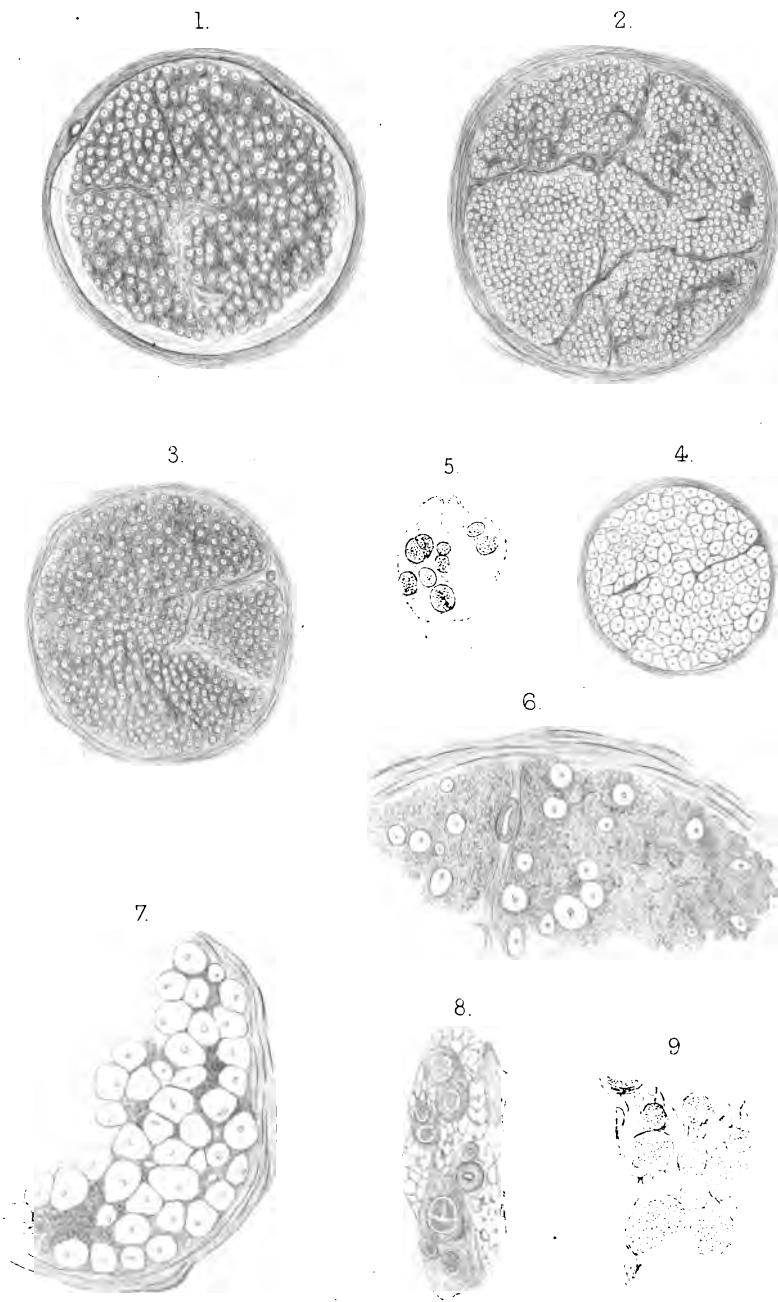
Figs. 5—8. Sections of motor and sensory nerves.

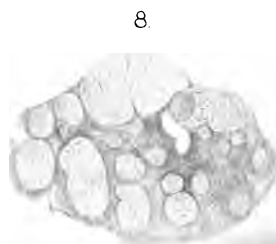
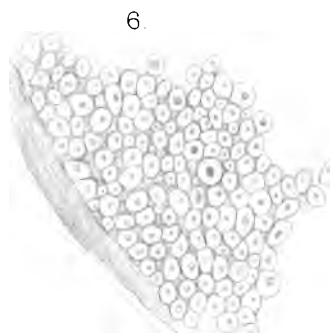
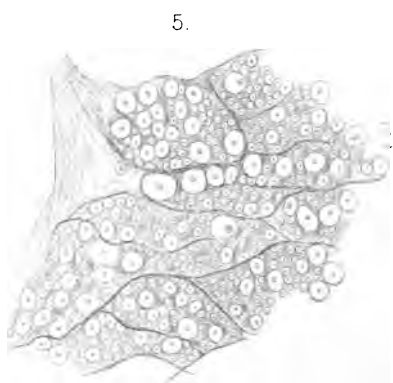
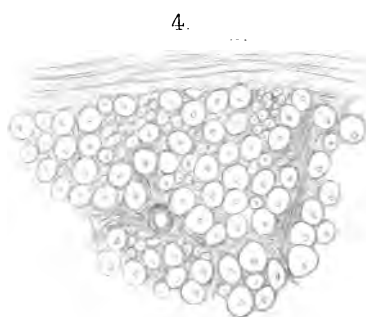
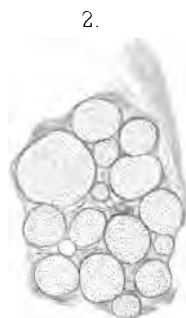
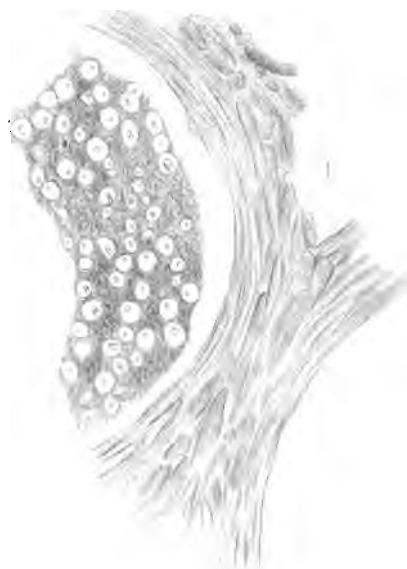
Figs. 5, 8. First division of 5th nerve; 5×250 , 8 with very low power (A. 4 in.).

6, 7. Facial nerve; $6 \times$ about 250, 7 with very low power (A. 4 in.).

PLATE VI.

Perforating ulcer of the foot.







ON
DELIGATION, FOR AORTIC ANEURISM,
OF THE
RIGHT CAROTID AND SUBCLAVIAN ARTERIES
WITH A NEW SPECIES OF LIGATURE.

BY
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(Received March 10th—Read May 27th, 1879.)

It will very probably be remembered by the Fellows of this Society, that I have already had the honour to bring to their notice four cases of double deligation of the carotid and subclavian vessels; and that of these four, three have proved successful. The operations were undertaken for the cure of innominate aneurism. I now beg to record a fifth case of such operation performed for a different object, viz. for an aortic aneurism. The vessels were tied with a material hitherto unknown in surgery. As the remarks, which I feel to be forced upon me by the peculiarities of the practice, must of necessity occupy some space, the report of the case will be condensed within the narrowest possible limits.

On 8th February, 1879, Dr. Green asked me to see with him John S—. The man was thirty-six years of age but looked

older, the scalp hair being quite grey. His father died of heart disease. There is no history of syphilis or of rheumatism. On the right side of the upper part of the chest I found strong pulsation extending over a considerable space, but not limited by any clearly defined boundary line. At the same part was incomplete dulness, with similar lack of distinct definition. Pressure symptoms were entirely confined to the right bronchus, that side of the chest being almost immobile, but very little air entering the right lung, while the left side was hyper-resonant, the respiration puerile. Great oppression with some pain referred to about the right nipple was complained of. There was no perceptible or sphygmographic difference between the radial pulses. Dr. Green had treated the case, by the best means known in medicine, since the 9th January, but the aneurism was evidently increasing, and Dr. Green kindly handed the case over to me. In the week after our consultation his symptoms increased, and at last the right chest was entirely immobile. On the 15th Feb. I tied the right carotid and subclavian vessels with a ligature to be described in the sequel. The man recovered easily from the immediate effects of the operation. The right arm remained cold for six hours. Almost immediately, the sensations of oppression and the pain about the chest began to diminish, the patient saying he felt well; shortly after, pressure upon the bronchus very much declined—air entering the small bronchi—there seemed, since vesicular murmurs were weak, to be some difficulty of expansion in the lung itself, the bronchus appearing free. He continued in a very satisfactory condition till 28th March, when some highly vexatious domestic incident greatly excited him; he insisted on getting up, threw himself about, and was with difficulty dissuaded from leaving the Hospital. A day or two after, a pulsation further toward the left was detected, this increased to the middle of April, then became stationary and then declined.

13th, May.—Some increased pulsation is still perceptible over the first bone of the sternum, and a little way to the right of this bone as low as the cartilage of the second rib. The symptom has, however, very much decreased since the beginning of the month. I have hopes, therefore, that

although the aorta, about the origin of the left subclavian, is dilated, a little further rest and treatment will improve this condition.

It should be noted that pulsation in the branches of the right carotid returned in a few hours, although none could be felt between the ligature and the bifurcation. Pulsation in the radial and brachial arteries was entirely absent for fifteen days. For another fortnight the radial pulse was only occasionally perceptible, and is even now very small. I have no doubt that, as in a previous case reported to this Society ('Med.-Chir. Trans.,' 1878), the whole subclavian vessel is obliterated.

The points of this case which require comment at my hands, are only two, but they are of very considerable importance, namely, the operation chosen, and the sort of ligature used. I will take them in that order.

The operation chosen.—As far as I am aware, no other case of aortic aneurism has ever been knowingly and wittingly treated by deligation of the right carotid artery, still less of that vessel together with the subclavian; I feel it, therefore, incumbent upon me to give my reasons for this choice. They are both theoretical and practical. In 1869, Dr. Cockle published some papers in the 'Lancet,' advocating in certain cases of aortic aneurism, deligation of the left common carotid artery. As far as I understand the author's able, but very condensed, remarks, he takes into account no other operation; at all events, his suggestions have been so acted upon by Mr. Heath, Mr. Holmes, and myself. In the cases of the two first-named surgeons, so much benefit resulted that the patients might practically be considered cured (Mr. Heath's patient suffered recurrence). In my own case, the disease was so far advanced that no cure, but merely prolongation of life and alleviation of symptoms could be hoped for; these followed in even a shorter time than was expected. But in considering certain points concerning the aortic arch, its branches, and the course of its blood-stream, I came to the conclusion

that distinctions must be drawn as to the operative treatment of aneurisms occupying different portions of that trunk.

Let me call attention to the mode of origin of the three great vessels, which has, I think, not attracted any attention. First, we see the great brachio-cephalic trunk, then, be it observed, there is no interval between that and the left carotid; there is not, as is usually figured and imagined, between these two vessels a bit of transverse aorta convex upwards, but a mere angle, a sort of V-shaped double septum. A little wider angle separates the left carotid from the subclavian. If the ascending aorta be severed from the rest a little to the proximal side of the innominate, and we look along the tube of the transverse part, we do not see the orifices of carotid and subclavian foreshortened into an oval or mere slit, as would be the case if these vessels were given off straight and plumb from the parent stem. On the contrary, these openings look nearly round, we seem to see right into the lumen of each branch, chiefly into the carotid. This results from the mode of origin of these large branches; they are not given off straight and rectangular from the transverse aorta, but, on the contrary, their roots take a very oblique direction to the left, and then swerve upward to assume each its proper course; hence the distal margin of each orifice lies on a level considerably lower than the proximal. Each such margin has running from it downwards, and to the right on each side wall of the aorta, a rounded ridge, so arranged that the projection of this distal lip, and two-fold spur—in shape not unlike a half funnel—catches the blood-stream as it courses along the main trunk and directs each its own share into its special branch. The arrangement of these ridges is such that they divide all the upper aspect, and a considerable part of the lateral surfaces of the aorta into districts, one for each vessel. Hence an aneurism, unless it spring from the lower wall of the transverse portion of the aorta, must almost of necessity belong to the district either of one branch or the other. Furthermore, there is no room on the upper surface of the arch between the innominate and left carotid for an

aortic aneurism ; such a tumour must belong to one or the other branch (see fig. 1).

FIG. 1.



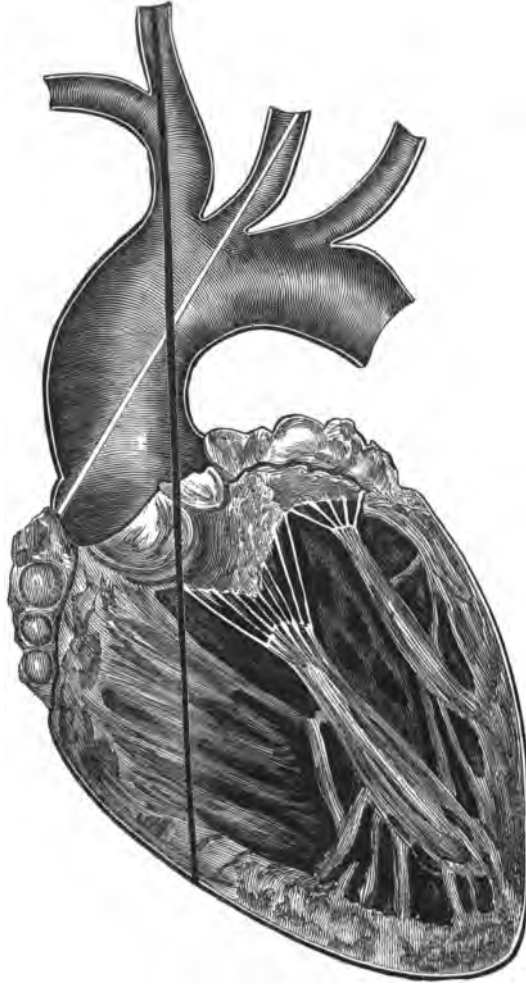
Arch of the aorta and large branches in section. It shows the obliquity of the roots of the great vessels and the ridges running from their orifices upon the aortic walls.

Now to another consideration. When a concretion is detached from an aortic valve it almost invariably passes into the left carotid, sometimes into the left subclavian, into the right carotid only about once in twenty-five cases. This circumstance has given rise to the assumption that the left carotid lies more fully than the right in the axis of the heart and ascending aorta: the very reverse is the fact. If a little hole be made in each carotid just below its bifurcation and a probe be passed along each common trunk, as far as it will go without the use of any force, and, if then, the front wall of the chest &c., be removed, and the ascending aorta

carefully opened, the two probes will be found crossing within the vessel. The right one will be seen to pass through the aortic opening not far from its left margin, and to lie well in the ventricle. The left one strikes the tendinous ring of the aortic orifice on the right aspect of the vessel. In most bodies the end of the instrument will be just within the heart, in others it will be in the sinus of Valsalva. The probe is never in the axis of the ascending aorta, but strikes the wall, be it of ventricle or of vessel, at a considerable angle. We must, therefore, attribute this preference of detached concretæ to some truer cause. Solids within a stream, if not too heavy, go with the strongest current; therefore, it would seem that some subtle curve or slope of surface directs the most potent rush of the stream somewhat obliquely across the ascending aorta in the direction of the left carotid. In this course the current would pass from the right aspect of the origin of the vessel, towards the left carotid orifice; it would occupy that portion of the trunk which in fig. 2, lies to the left of the white probe. Probably the potent flow of blood in this direction and into the left carotid, is intimately associated with the preponderance of the left brain and of the right half of the body.

Now, if this be the more rapid part of the current, it follows that outside it, to its right there must be a slower stream—and it is easily comprehensible, that if the left carotid be occluded the force of the rapid current must be diverted towards the right, whereby the normally placid stream will be disturbed and rendered more turbulent. Hence it would appear that if aortic aneurism is to be treated by deligation at all, we must, as far as our means of diagnosis will allow, carefully distinguish between such as arise from one or the other portion of the aorta. I am, of course, well aware that in many instances such diagnosis is difficult—also that often the extent of vessel involved is so great that no such distinction exists, the disease over-stepping the boundary limits and in such cases the propriety of any operation is, to say the least, doubtful. But I also know by the teachings of much careful experience, that when the aneurism springs

FIG. 2.



from a fairly limited portion of the arch, observation as to the signs of pressure on the right bronchus or on the trachea, as to the tracing of the sphymograph, as to the particular veins chiefly or alone congested as to the place at which the tumour makes its appearance, as to the condition of the voice

and larynx, as to the place and direction of neuralgic pains, &c., indicate with very considerable precision the site of the disease. This has certainly been the fact in many cases that I have watched with minute care. The disease of John S. for instance, presented no diagnostic difficulties: the absence of venous congestion, or of any observable difference between the two pulses, the wide-spread character of the very considerable pulsation and its undefined boundary over the front of the chest, the perfectly free entrance of air to the left lung with almost entire occlusion of the right bronchus, defined the situation of the tumour with certainty, as being the back wall of the ascending aorta near the first angle and not far from the orifice of the brachio-cephalic artery. It belonged to the innominate, or, as I prefer to call it, the right aortic district. The two districts, right and left to the one or other of which, all aortic aneurisms amenable to operative treatment must be taken to belong, are probably separated from each other by a somewhat curved line running from the outer or right side of the aortic opening to the left carotid orifice. Thus to the right system will belong the right wall of the ascending aorta, and on the back and front wall a semilune, one of whose horns is at the outer valve, the other at the left carotid opening. Any operation for an aneurism in this district should be on the right vessels. The left district includes the left wall, a considerable adjoining space on the front and back of the ascending aorta, and all the transverse aorta from the proximal margin of the left carotid opening. Taking these considerations all together and applying them to the case of John S., it is evident that had I tied the left carotid instead of the right vessels I should have diverted the rapid current just described, and most probably to the right side of the parent stem, in other words toward the aneurism; and I should have injured rather than benefited my patient, whereas by occlusion of the innominate artery the quiet stream was rendered still more placid, and coagulation facilitated. The result has fully justified these considerations and even the symptoms of dilatation of the aorta in the distal part of the arch, help to prove this view. The

aorta at this part was not strong enough to bear the additional strain of an augmented stream especially when excitement was superadded.

If it be objected to me that all this about aortic currents is mere theory, I reply that I can imagine no means, whereby the presence or absence of differing rates of flow within the vessel could be absolutely demonstrated, that even if we could remove a living chest-wall and render the aorta transparent, we still could only see the relative force in different parts of the stream by watching the behaviour of floating solids. Nature often performs for us that experiment with the results just stated.

Nevertheless, I hold that theory alone hardly offers sufficiently solid foundation for an operation of this magnitude unless supported by some facts in practice. In the Museum of the College of Surgeons (Pathological series No. 1596A), is a dissected specimen taken from the woman upon whom in 1865 Mr. Heath performed deligation of the carotid and subclavian. As is well known, the diagnosis was inaccurate the aneurism being not innominate but aortic, solidification by coagulum took place, and be it observed the tumour occupies the right side of the ascending aorta—just that part which lies to the right of the white probe in fig. 2. Again, one of the patients whose case I reported here last December, **Laura G—**, had aorto-innominate aneurism, the aortic part being situated on the right front wall of that vessel. I tied the right carotid and subclavian. The patient is well and has lately married. Upon these practical illustrations of my views I founded the choice of operation.

Ligature used.—The deligation of large vessels near the heart is a far more fatal operation than the tying of arteries at a greater distance from the centre, and very much of this increased mortality is due to secondary hæmorrhage, indeed, this has been the invariable result of tying the innominate or the first part of the subclavian, therefore deligation with any ligature now in use of those vessels and of certain others somewhat similarly circumstanced, cannot in my opinion be justified. Mr. Erichsen in his work on surgery, gives fourteen

cases of each operation, all, I believe, that are on record. Of the innominate cases one bled, but was saved, two died before the period for bleeding arrived, eleven died of hæmorrhage. Of the fourteen subclavian cases, twelve bled to death, the others succumbed before the period of hæmorrhage. Although deligation of other arteries exposes the patient to less danger, according to their anatomical peculiarities, yet always there is some risk from this cause. In all the above twenty-eight cases the ligature used was silk or twine, that is, a material which divides the two inner coats, and then must come away by ulceration and sloughing of the outer tunic of the artery. It is plain that if under these conditions certain circumstances occur, still more if they concur, bleeding from one or other of the severed ends must inevitably result. One such circumstance, always present when the innominate, first part of the subclavian, or lower part of the carotid, is tied, is the neighbourhood of a large branch or vicinity of the ligature to the trunk, whence the vessel tied is derived. Moreover, the use of an insoluble ligature promotes suppuration of the wound and softening of the arterial coats. By the use of carbolised catgut combined with any means that shall most certainly secure primary union we avoid some of these dangers, but not all of them; as evidenced by the fact that, even when all these means have been employed, secondary hæmorrhage does sometimes take place. I believe this bleeding occurs because, either with the intention of the operator or unavoidably and against his will, the ligature cuts through the two inner arterial coats and may produce ulceration of the outer one,¹ or even if the outer coat preserve its continuity, being now exposed unsupported to the whole force of the blood-stream, it may expand into an aneurism at the seat of ligature.²

A few years ago, I asked myself whether the organisable or soluble ligature would permit us with safety to dispense with the division of the inner arterial tunics, this dangerous

¹ See Mr. Bryant's cases, 'Clin. Soc. Trans.,' vol. xi, p. 1. Mr. MacCarthy's case and Mr. G. G. Heath's (Newcastle) case, 'Lancet,' vol. i, 1877.

² Mr. T. Smith's case, 'Clin. Soc. Trans.,' vol. xi, p. 52.

element in deligation, and on the 10th December, 1878, when I brought to the notice of this Society three cases of double distal ligature for innominate aneurism, I alluded at some length to this subject, rather with the view of eliciting opinions, than of establishing a practice. I confess to a feeling of surprise on finding that two surgeons whose names are well known in connection with aneurism, still adhere to the view that organisable ligatures should be tied so as to divide the coats of the vessels, thus, exposing patients to those great dangers which I have just shown to be inseparable from such practice. Probably a belief, perfectly well founded, in the unreliability of catgut, and a feeling that it is very difficult to tie that material tight enough to be firm, yet loose enough to leave intact the vascular coats, lie at the root of these opinions. Nevertheless, it is simply obvious, that if we had a material possessing a fixed rate of organisation, which would not divide the arterial coats, we could cast around the vessel a permanent band, that should effectually stop circulation in the artery, yet leave its walls entire and hæmorrhage-proof.

The unreliable nature of catgut lies in the fact that under like conditions its conduct is various; we cannot tell what any specimen will do; nay, different parts of the same cord will act differently. One piece of the material will be dissolved in from forty-eight to sixty hours; another is found entire after a fortnight. Sometimes, though rarely, a piece is not absorbed at all, sometimes it seems to divide all the arterial coats very quickly; in other instances it pierces a hole in them. At first I was inclined to attribute this untrustiness to the varying periods, during which the catgut was kept in antiseptic oil, and I used to carbolise my own ligatures with somewhat better, but still imperfect result. I then inquired into the process of the first manufacture, the making of catgut itself. I believe it is from these processes, that the unreliable nature of our antiseptic ligatures is derived. The following is slightly abbreviated from Chambers' Cyclopædia. "Catgut is made from the intestines of sheep, rarely from those of horses, mules, or asses, never from

cats. After cleaning the parts from feculent and other impurities, detaching mesenteric fat &c., the gut is steeped in water for several days, until the external membrane (probably the peritoneum) can be scraped off with a blunt knife. After another process of soaking and scraping, the small intestines are separated from the large. These latter are used for other purposes, while the former are returned to a solution of potash, carbonate of potash and a little alum, they are lastly drawn through a perforated brass thimble and assorted into their various sizes. They are then subjected to the fumes of burning sulphur, which acts as an antiseptic and arrests decomposition.

After reading this and observing that putrefaction in water plays a large part in the manufacture of catgut, I no longer wondered at its variability; for it surely must happen that one piece, even one part of a cord must have been, previous to fumigation, more rotten and putrescent than another.

But even this was to my mind less cause of objection to catgut as a ligature in continuity than the form of the material. I felt that a round cord could hardly be relied upon not to cut arterial coats, and I was determined that my ligature should be flat, a form with which I found by experiment arterial coats cannot be divided. Many materials were thought of, a good number made the subject of experiment; but all rejected until the idea occurred to me that the most fitting substance with which to surround an artery would be arterial tissue. I procured with some difficulty the aorta of some oxen in a perfectly fresh state, and placed them in a three per cent. solution of carbolic acid. Without troubling the Society with a description of my failures I may say that after a little time I succeeded in separating the middle from the outer coat, and, by cutting the former spirally round and round, in procuring flat tape-like ligatures or bands of much more than the requisite length. But such a ligature is, though very strong, much too elastic to bear a reliable knot. This superfluous elasticity can be removed by suspending the cord and hanging on it a weight of from one to three pounds (according to the breadth, &c., of the piece) so as to stretch it to at

least an additional half of its length ; a piece of six feet should stretch to nine, of four feet to six, and so on. Thus treated, the material dries into a horny or vellum-like substance, which I store in antiseptic gauze, and place ten minutes before use in carbolic solution when it again becomes extremely soft and pliable. Some experiments were tried, not on the human body, whereby I found that on the thirteenth day after burying a piece of this middle coat about $1\frac{1}{2}$ inch square between the abdominal muscles and skin, the material was still *in situ* and but little changed ; it was living ; it had, as it were, simply come to life. Some other issues were also tried, which for obvious reasons I cannot now describe. I then tested the security of the knot, and its power of resisting an expansile force, by stretching a tolerably thick india-rubber cord until it was quite thin and tying two ligatures tightly round it. The diameter of the tied portions was measured and the cord released ; the constricted parts had but very slightly dilated. The cord was then cut between the two ties, one portion placed in glycerine, the other allowed to dry. On the fourth day the wet one had perhaps a little increased in diameter, the dry one had plainly diminished, facts attributable to the soaking and desiccation respectively. The knots had not yielded in the least ; it ought perhaps to be said that I think the security of the knots depends upon considerable tightness of its second part or involution—for this material when pulled upon gets narrower and when released resumes its former breadth ; the broadened part forming a sort of buttress against the tie of the narrowed portion entirely prevents slipping.

My last experiment was the case just narrated, which indeed chanced to form the meeting point of two enterprises ; it has by furnishing proof of the first mentioned views enabled me, I think, to educe correct indications for tying one or the other set of vessels in different forms of aortic aneurism, whereby a precision hitherto wanting will have been imparted to our practice. Also although one case cannot prove invariable success, it has enabled me to introduce a ligature whose

mode of occluding the vessel is such that it will, I trust, eliminate from this branch of surgery secondary hæmorrhage at the place of deligation, even when such arteries as the first part of the subclavian or the innominate are tied. If this be so, and the view is not I believe too sanguine, a wider field will have been opened to surgery and a high degree of safety conferred on a number of operations, which hitherto have proved all but uniformly fatal.

ON THE
PATHOLOGY OF LUPUS,
WITH
SPECIAL REFERENCE TO THE APPEARANCES
DESCRIBED AS GIANT CELLS.

BY
GEORGE THIN, M.D.

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SUCH portion of our knowledge of lupus as is established beyond controversy both as to the facts and the theories explaining them is due to the investigations of Auspitz and Virchow. The former observer in a memoir on the cell-infiltrations of the corium, published in the Vienna Jahrbücher for 1864, sums up his views regarding the pathology of the disease, by stating that it is essentially a cell-infiltration which penetrates the corium uniformly throughout its entire depth. Beginning in the form of nodules which glimmer through the moderately swelled epidermis, it produces elevations which can usually be felt, although in some cases they appear as simple maculæ. Later, these nodules extend until they join each other, and form an extensive area of infiltration, whilst fresh nodules are formed at the periphery of the affected part.

Virchow in his work on Tumours, gives a similar account of the process, describing the cell-masses which are formed

as granulation-tissue, and on that account bringing lupus into the same category with leprosy.

The next work on lupus of importance is the memoir published by Friedländer in Virchow's Archiv in 1874. This observer called attention to the existence of nodules of a special kind, in the midst of the granulation tissue. The cells in the nodules are, he found, larger than the cells of the granulation-tissue, although their nuclei are about equal to those of the latter cells. The diameter of the nodule-cell is three times that of the granulation-cell, and the largest of these nodule-cells, pass by transition direct into giant cells, which are invariably to be found in the nodules. These giant cells are, he tells us, somewhat spherical structures, provided with delicate processes which taper to fine points. Their protoplasm has the same characteristics as that of the nodule cells; their nuclei are also similar, except that they are frequently oval instead of round, especially in the larger forms. The number of nuclei is always considerable and may exceed a hundred.

The importance of this description is best understood by taking it in connection with Schüppel's¹ investigations on tuberculosis of the lymphatic glands. "I distinguish in tubercle of the lymphatic glands," remarks this author, "three different forms of cells, namely, giant cells, large epithelioid cells, and small lymphoid round cells." Friedländer states that the giant cell probably never fails to be present in the lupus nodule; Schüppel that it is scarcely ever missed in tubercle. I have only further to add that a comparison of Friedländer's and Schüppel's plates shows that they had before them elements of an exactly similar nature.

Since the publication of Friedländer's memoir most of the investigators who have written on lupus, have confirmed his observations regarding giant cells. Professor Lang² of Innsbruck, forms, however, an exception. He observed appearances similar to those described by Friedländer, but

¹ 'Untersuchungen ueber Lymphdrüsen-Tuberkulose,' von Dr. Oscar Schüppel, Tübingen, 1871.

² "Zur Histologie des Lupus," 'Archiv für Dermatologie u. Syphilis,' 1875.

considers them to be degenerated sweat tubes and ducts. Stilling,¹ the latest German writer on the subject, so far as I know, believes that some of the appearances are, as Lang has advanced, due to degenerating epithelial structures, but maintains that the true giant cell also exists in lupus, and he figures examples similar to those drawn by Friedländer. Thoma, the author of an exhaustive and very valuable memoir on the pathology of lupus, in 'Virchow's Archiv,' in 1875, expresses his belief that all the cells of the lupus-infiltration are probably derived from the colourless blood-cells found in the perivascular spaces, these forming successively, in different stages of development, the granulation cell, the epithelioid cell, and the giant cell.

Although the discovery of giant cells during the last few years in very different kinds of diseased tissues has in one sense somewhat diminished the importance of Friedländer's observations, the interest attaching to the question is from another point of view only increased. As the "giant cell" is so frequently met by pathologists in whatever direction they are exploring, often turning up where least expected, further knowledge regarding this equivocal structure has become one of the pressing needs of medical science.

The results of an examination of lupus tissue, for which I am indebted to the kindness of Mr. Morratt Baker, have proved interesting chiefly in relation to this question of giant cells. A careful analysis of these objects, in their different stages and forms, has led me to form the conclusion that the giant cells of lupus are not independent organic structures, but that, on the contrary, the wall of the so-called cell is the altered coat of a diseased blood-vessel, that the appearance described as protoplasm is produced by a disintegration of red blood-corpuscles, that the nuclei on the periphery are nuclei of the vascular walls, and that the nuclei of the centre, when they are present, are the nuclei of colourless blood-corpuscles.

¹ "Einige Beobachtungen zur Anatomie und Pathologie des Lupus," von Dr. Med. Heinrich Stilling, 'Deutsche Zeitschrift für Chirurgie,' Band viii, 1877.

Before, however, entering on this, the main subject of my communication, I shall state briefly the evidence which is furnished by the case I have examined regarding some other points in the pathology of lupus which are still *sub judice*.

The lupus tissue was procured from a boy, set. 9, who was under Mr. Baker's care in St. Bartholomew's Hospital. He had an isolated patch of lupus on the anterior surface of the left thigh, measuring about two inches in its long diameter; it had a raised border and was nowhere ulcerated. There was also a small lupus tubercle on the skin of the penis. Both patches were excised by Mr. Baker. I examined part of the patch from the thigh and sections of the whole of the tubercle from the penis.

The distribution of the cell infiltration in this case corresponds exactly with that found by Thoma in a number of cases which he examined. In the apparently sound skin, for a considerable distance beyond the developed disease, the superficial blood-vessels of the corium are invested with a thin covering of cell-infiltration. There is no cell-infiltration here between the bundles of the connective tissue. Coming nearer the extensively diseased area the external root-sheaths of the hairs and the sebaceous glands are found disintegrated and their place occupied by a mass of cell-infiltration from the vessels of the hair-follicle, the surrounding connective tissue being still unaffected. Approaching the centre of disease we find nodules of cell-infiltration developing around the blood-vessels and displacing the connective tissue, until finally we come to a point where the papillary layer of the skin has completely disappeared, its place being occupied by a large mass of cell-infiltration, in which the areas of the nodules, by whose coalescence it has been formed, can mostly still be recognised.

Deeper in the corium, and mostly separated from these superficial masses by unaltered bundles of the *pars reticularis*, we find large oval or round nodules of cell-infiltration, and by examining these deeper nodules in different stages it is seen that the seat of disease here is the territory of a sweat coil. It has been stated by Lang that in lupus the sweat

glands take on new growth. In this case it has certainly not been so. The cell-infiltration begins around the vessels of the coil, and the glandular epithelium simply passes from one stage of disintegration to another.

Localised nodules had also begun to form amongst some of the superficial flat vesicles.

The appearances which I have described support the views of those observers who describe lupus as a cell-infiltration which spreads outwards from the blood-vessels.

I have found no evidence in favour of Lang's opinion that the cell-infiltration originates in a growth of the protoplasm of the capillary wall, nor anything to support the conjecture that there is proliferation of the connective-tissue cells. The explanation given by Auspitz and Virchow of the connective-tissue origin of the cell-infiltration was advanced at a time when the presence of several nuclei in any space between the bundles was believed to afford proof of cell-proliferation. Since that period the discovery that in morbid conditions the colourless blood-corpuscles enter the interfascicular spaces, and the better knowledge we now possess regarding the nature of connective-tissue cells, have revolutionised our conceptions regarding the origin of cell-infiltration.

As a matter of fact the connective-tissue cells of the corium are exceedingly difficult of demonstration, and I know of no text-book in which an attempt is made to show more than that there are cell-nuclei on the bundles. Any endeavour, therefore, to discuss assumed morbid changes in these cells is premature.

I agree with Thoma in thinking that the size of the cells in the lupus-infiltration, the positions in which they are first found, and, I may add, their behaviour under staining agents, concur to render it very probable that they are colourless blood-cells undergoing various stages of development and degeneration.

Scattered through the lupus growth, and more especially situated (generally excentrically) in the nodules that compose it, are the structures that will now engage our attention—the "giant cells" of Schüppel and Friedländer. I find in my

preparations numerous examples of appearances identical with the giant cells figured by these and subsequent observers. This identity will be apparent to any one who compares my figures with theirs (see, *e. g.* figs. 3 and 5).

The appearances in question can be made very apparent by a particular mode of preparation. The dye eosin has a special elective affinity for some tissues, red blood-corpuscles and blood-vessels sharing with horny epidermis and striped and unstriped muscle this preferential selection. The giant cells of lupus stain as deeply by eosin as blood-corpuscles or muscular fibre do, and by judicious manipulation with the dye a section of lupus tissue can be made to show these structures coloured a deep orange red in an almost colourless ground. Subsequent staining by logwood brings out the nuclear elements present in the mass.

The fully-developed giant cell is usually seen in section as a circular or oval mass with a well-defined contour, which, for the purposes of description, I shall term its wall (fig. 3 *a*, fig. 5 *a*, fig. 12). The mass is granular, and, as I have already remarked, stains deeply by eosin. The wall is generally lined on the inner surface, more or less completely, by nuclei. In the substance of the mass, there are frequently no nuclei, or they may be present sparsely or in great numbers in one part and not in another, or be found only in the periphery, or only in the centre. From the bounding wall of the mass process-like prolongations pass outwards into the tissues which surround it (fig. 3 *a*). Sometimes there is a space between the mass and the surrounding tissues, and the prolongations are seen very distinctly crossing it. When this space exists oval nuclei, which have the character of the nuclei of connective-tissue cells, are found on both its bounding walls. But from this typical giant cell there are many departures. Oblong membranous-looking patches, long narrow strips, some of them very small, others very large, are found, more or less granular, with more or fewer nuclei, and staining exactly like the giant cell (fig. 5, *c*, *d*).

Further, if we search for the early stage of development of

the giant cell in parts of the healthy or comparatively healthy tissue adjoining the lupus infiltration, we find the following transition stages:—Giant cells are found in which the contents of the cell are homogeneous at one part and granular at another (fig. 7); others in which the contents are entirely homogeneous, whilst the wall and processes fulfil the characteristic requirements of the giant cell, the nuclei, in this case, being limited to the wall, but still differing in no respect from the nuclei on the wall of the granular giant cell. In some of these cells with homogeneous contents the likeness of the whole structure to a blood-vessel becomes apparent, and occasionally the wall is found to show the characteristic muscular coat of an artery. Going another stage backwards, we find a giant cell with homogeneous contents and red blood-corpuscles in the centre of the homogeneous mass—sometimes in considerable numbers—the wall and its relations still answering to the requirements of the giant cell (fig. 6). In all these cases the characteristic staining by eosin is maintained, and the conclusion becomes forced on us that the peculiar staining of the granular mass in the typical giant cell is due to the substance stained deriving its origin from the red blood-corpuscles.

In other sections of giant cells we find the coats of the original blood-vessels still represented, the inner coat being represented by the nuclei of its endothelium, and the middle and outer coats by considerable thicknesses of characteristic tissue (fig. 9).

When we find a giant cell bounded by a single wall, which is fringed on its inner surface with a line of nuclei, the resemblance of these nuclei to those of the vascular endothelium must strike every one. Similarly, in sections in which there is considerable thickness of wall a number of elongated, narrow nuclei are found in it, which I believe to be equally characteristic of the coats of the blood-vessels, of the size of the larger vessels usually found in the corium.

It occurred to me that if the so-called giant cells are in reality disintegrated blood-corpuscles in degenerated blood-vessels, it should be possible to isolate the mass in some

length. And this I have succeeded in doing. Figs. 10 and 11 are drawings of an elongated, hollow, granular mass thus isolated, the shape affording evidence that it is the cast of a dilated blood-vessel. This mass consisted of a finely granular substance, which stained by eosin precisely the same tint produced by the dye on the granular substance of the giant cells, and in other respects the similarity was complete. Embedded through the whole thickness of the mass scattered colourless blood-cells were observed. On the edge of the cast, and on its external surface, formed elements were found at several points. These were not granular, and were probably detached portions of the wall of the vessel, which had remained adherent to the granular contents. I found no other elements in my preparations except the so-called giant cells, whose characters corresponded to the mass I have just described.

I have also figured part of a preparation in which a group of giant cells was found in one microscopic area of the nodule from the penis (figs. 4 and 5). On studying this group it became evident that all the structures composing it belonged to one or two blood-vessels. Between two of the giant cells blocks of a substance containing nuclei, and coloured orange red by eosin, but not granular, connected one cell with another. By careful regulation of the fine adjustment it was seen that the giant cells corresponded to parts of the vessel in which the wall and its contents had been cut transversely or obliquely, and that the non-granular communicating links consisted of longitudinal sections of the wall of the vessel alone.

I think it is probable that the formation of this appearance in the vessels is attended by great distension of the diseased wall. In a thick section I have several times been able by using the adjustment to follow a giant cell of considerable size into a comparatively small vessel without contents. My experience on this point is, however, not sufficiently large to warrant the expression of a definite opinion, and in these cases I may have been misled by a branch of the diseased vessel.

Friedländer has described the giant cell as a constant element in the minute nodule, the presence of this cell being relied on as evidence of the relationship of lupus and tubercle. The outlines of these nodules are distinct in the preparations I have studied (figs. 2 and 3). Instead of the expression giant cell, however, I substitute the term diseased blood-vessel. In many of these nodules the blood-vessel from which the limited cell infiltration has taken its origin still remains recognisable, but mostly it is found as a typical giant cell. The area of the nodule is often bounded by a distinct layer of fibrillated connective tissue (fig. 3, *b*,) and a comparison of areas in different stages of development leads me to infer that this connective tissue represents the adventitia of the vessel. The effusion from the vessel detaches the adventitia from the other coats and pushes it outwards, the gradually enlarging area in which the cells of the nodule are observed being thus formed.

Such a growth of the adventitia as is implied in the formation of a bounding wall of the area must be, of course, considerable. It has been observed and described by Thoma, who compares the layers of new growth in extreme cases to the membranes of the Pacinian corpuscle.

In eosin-stained preparations the nodules take on the dye more strongly than the cell-infiltration surrounding them, indicating the existence there of some substance which is not present in parts where the diseased process has not reached the same degree of specific change.

The process-like fibres which in many cases pass from the giant cell into the surrounding tissues (fig. 3), and which have been described as processes of the cell, answer to the tests of connective-tissue fibres. They have the appearance and position of fibres which in healthy blood-vessels connect the vessel with the connective tissue in which it lies, and are, I believe, specially related to the adventitia.

Having completed this analysis of the "giant cells" in lupus, I will, in conclusion, permit myself a few general remarks on the subject of the diagnosis of cells in so far as it refers to giant cells. In lymphoid and some other cells a

more or less granular appearance is detected by the microscope. Hence has arisen a natural tendency to associate any granular substance in which a nucleus or nuclei are embedded with the conception of a cell. If this substance is in a mass, and if many nuclei are embedded in it, the difficulty is surmounted by the conception of giant cells. The correctness of this mode of reasoning depends on the accuracy of the assumption which really underlies it, namely, that degenerating tissues do not become granular, and that nuclei may not be easily demonstrated in them after every trace of the cell-substance proper has disappeared. Both these assumptions are contradicted by facts.

The conclusion at which I have arrived has some analogy with that to which M. Cornil¹ has lately come after an examination of tubercular lymphatic glands. This pathologist states "that the giant cells of Schüppel and German authors always develop in the interior of the vessels after the circulation in them has been arrested, when the lymph-corpuscles or endothelial cells accumulated in the lumen of the vessel continue to live and to grow at the expense of the fibrin and red corpuscles which are in contact with them." The foci or areas found surrounding one of these "giant cells" in tubercle are simply, he believes, "the contents of a small obliterated vessel whose wall has been destroyed."

The points of difference between my views and those of M. Cornil are that I find no evidence of growth in the endothelium of the vessel, and no reason to believe that the lymph-corpuscles are nourished and multiply at the expense of the red corpuscles.

Lang² has described and figured diseased blood-vessels in lupus, identifying the morbid change as similar to that described by O. Weber under the term vitreous swelling. "The vascular elements, he observes, swell, becoming shining, stiff, and brittle. Such a vessel gives the impression of a hard tube, the intima appearing splintered, whilst the cellular elements are distended and glassy, and the connective tissue acquires an unyielding or uniformly opaque aspect."

This morbid appearance has been so fully described and

¹ Robin, 'Journal de l'Anatomie,' No. 3, 1878.

² Op. cit.

figured by the author I have just quoted that I shall not report in detail the examples of it which I have observed. I shall content myself with remarking that I have both isolated and observed in sections examples of blood-vessels in which the walls had undergone this transformation, and that I regard it as an early stage in the process that leads to the formation of the "giant cells" and the nodule which forms around the latter.

The term giant cell is, it may be fairly contended, an unfortunate one, inasmuch as it not only prejudices the nature of an appearance whose origin has not been sufficiently traced, but it has grouped together structures which are manifestly dissimilar. If the giant cell of lupus is compared with the *myéloplâque* of growing bone, which is also described as a giant cell, it is impossible not to be struck by the points of difference, even more than by the points of similarity. This diversity has been insisted on by Professor Charcot, who¹ has recently stated "that the giant cells of tubercle must be held as absolutely distinct from similar elements found in other neoplastic growths, as sarcomata, granulations, inflammatory exudations of serous membranes, &c." The first are, he tells us, multipolar, with branching processes and peripheral nuclei, whilst the second have none or few processes, and their nuclei are scattered in the body of the cell, and not at its periphery. This difference applies equally if we substitute the giant cell of lupus for that of tubercle, and its explanation will be found in the observations which I have laid before the Society. When the giant cell is produced by a diseased blood-vessel with disintegrated contents, the adventitia supply the multipolar processes, and the nuclei of the endothelium of the intima the peripheral nuclei. Into the nature of the other form of giant cells, of which the *myéloplâque* of Robin may be taken as the type, it is beyond the scope of this paper to enter. It is distinguished by the absence of the special features which would connect it with a blood-vessel.

¹ Quoted by Malassez et Ch. Monod, 'Archives de Physiologie,' 1878, No. 4, p. 398.

That in diseases which clinically differ so much as tuberculosis and lupus a characteristic morbid product, like the "giant cell," should form so conspicuous an element in both is undoubtedly a fact of great interest, but it leaves the question of the identity of the two morbid conditions untouched. Assuming the correctness of my interpretation, the cause of the disease of the blood-vessels in the two instances may be widely different, and there is in each of them amorphous morbid material in the affected areas, which may have much to do with the extension and virulence of the maladies, but regarding which the microscope affords us little or no information.

So far as positive evidence justifies an opinion, the case of lupus with which I have been dealing is a case primarily of disease of the walls of the blood-vessels. The cell-exudation, the molecular disintegration of the fibrillated connective tissue, and the mode of extension of the disease, are all explicable by the degeneration of the vascular coats. This opinion is not incompatible with any of the facts contributed by previous observers; on the contrary, it is supported by them.

EXPLANATION OF PLATES VII AND VIII.

(Pathology of Lupus. Dr. George Thin.)

FIG. 1.—Vertical section through the hypertrophic edge into the apparently healthy tissue. *a*, Epidermis; *b*, large mass of cell-infiltration produced by the coalescence of nodules; *c*, hairs; *d*, a sebaceous gland (seen by a higher magnifying power to be degenerated and filled with exudation cells); *e*, a sweat coil (seen by a higher power to be infiltrated by cell-exudation; the upper part of the coil is disintegrated, the lower part is little changed); *f*, cell-infiltration occupying the position of the external root-sheath of a hair. At this part the epidermic cells are almost entirely destroyed; the upper part of the root-sheath (indicated in the drawing) is unchanged. *g*, Cell-infiltration limited to the perivascular space in the vessels of the corium; *h*, fat; *i*, an artery. $\times 8$.

FIG. 2.—*a*, One of the nodules or areas from which the lupus growth extends (several nodules are still recognisable in the mass; the black marks within them are "giant cells" or degenerated blood-vessels; all the dark objects in the figure, except the two hairs marked *b*, are giant cells); *b*, hairs; *c*, connective tissue of the corium; *d*, fat; *e*, cell-infiltration. $\times 14$.

FIG. 3.—Giant cell with its nodule (marked *a* in Fig. 2). *a*, The "giant cell;" *b*, fibrillated connective tissue bounding the nodule; *c*, one of several small blood-vessels which can still be traced in the nodule. $\times 260$.

FIG. 4.—About one half of the small lupus patch excised from the penis. *a*, Epidermis; *b*, connective tissue of the corium; *c*, lupus cell-infiltration; *d*, a giant cell (*a* in Fig. 5), forming one of several which make up the group shown in Fig. 5 (all the darkly marked objects in the cell-infiltration, except *e*, are either typical giant cells or parts of the coats of diseased blood-vessels in which the granular appearance is not present); *e*, section of a small hair; *f*, two large giant cells of typical appearance when seen under a high power (from their position probably parts of an artery and vein.) $\times 30$.

FIG. 5.—The group shown in Fig. 4 more highly magnified. *a a'*, At one focus typical giant cells; on changing the focus the appearance is found to be continuous with the vessels shown in the figure); *b*, "giant" cell, evidently part of the vessel next it, marked *a'*; *c*, longitudinal sections of the degenerated walls of blood-vessels. $\times 260$.

FIG. 6.—Section of a vessel showing the first stage of formation of a giant cell. *a*, Homogeneous coagulum; *b*, still unaltered red corpuscles; *c*, nuclei of the endothelium of the lining membrane of the vessel; *d*, normal connective tissue next the vessel; *e*, cell-infiltration appearing between the wall of the vessel and the adjacent bundles of connective tissue. $\times 260$.

FIG. 7.—Section of a vessel showing a more advanced stage towards the formation of a "giant cell." *a*, Homogeneous coagulum; *b*, nuclei of the intima; *c*, granular matter beginning to form in the coagulum; *d*, healthy connective tissue. $\times 260$.

FIG. 8.—Giant-cell formation developing whilst some unchanged red corpuscles are still found in the coagulum. *a*, Homogeneous part of the coagulum; *b*, granular part; *c*, unchanged red blood-corpuscles; *d*, normal connective tissue; *e*, small blood-vessels, with formation of lupus cell-infiltration around them. $\times 260$.

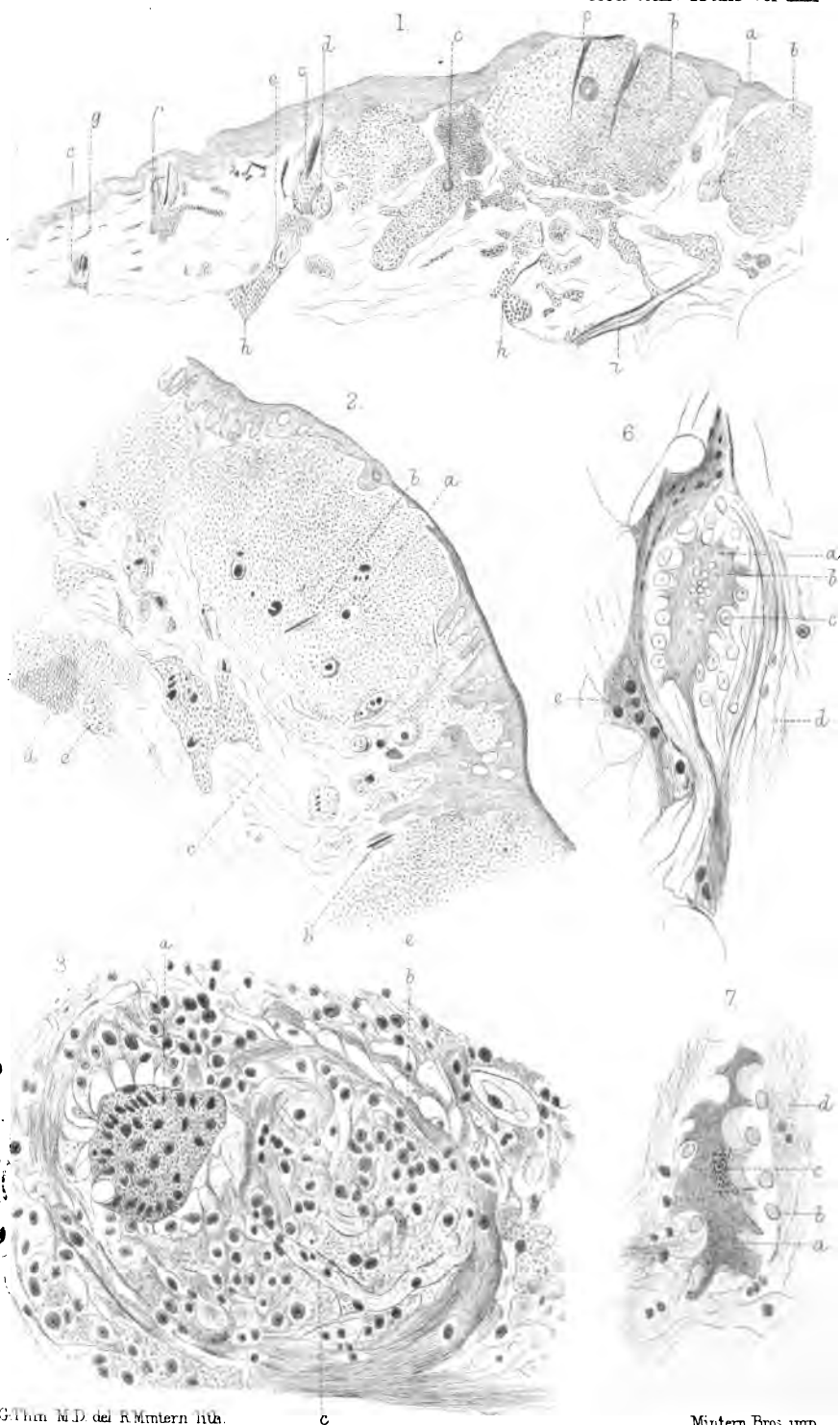
FIG. 9.—"Giant cell," in which the walls of the vessel, *a* and *b*, are still distinguishable. $\times 260$.

FIG. 10.—Cast of vessel isolated from lupus tissue, and showing all the characteristics of the supposed giant cells. $\times 45$.

FIG. 11.—Part of the cast represented in Fig. 10, more highly magnified to show the granular formation and the colourless blood-cells throughout it; *a* and *b*, adherent elements of the blood-vessel. $\times 260$.

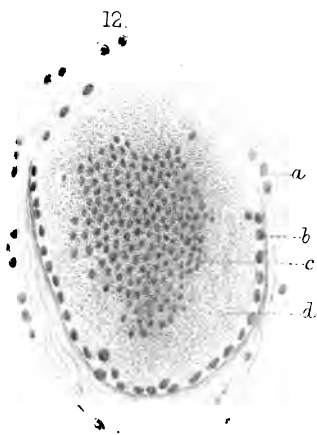
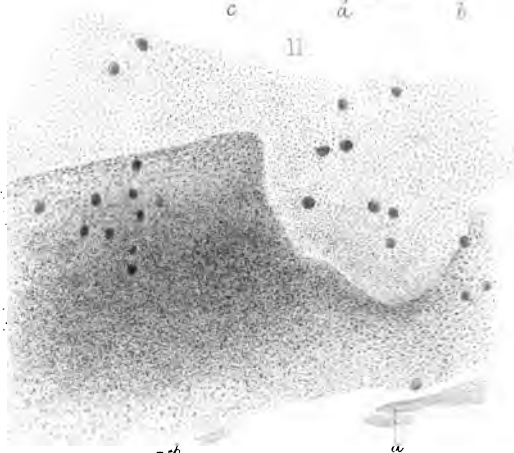
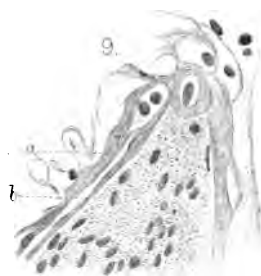
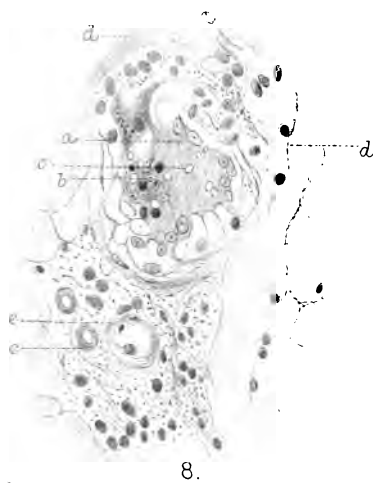
FIG 12.—Giant cell. *a*, colourless blood-corpuscles in connective tissue; *b*, nuclei of the endothelium; *c*, mass of colourless blood-corpuscles accumulated in the centre; *d*, granular product of disintegration of red blood-corpuscles, so-called "protoplasma."

(All the figures have been drawn to scale by camera lucida.)



G. Thun N.D. del. R. Mittern lith.

Mittern Bros imp



G. Thos. M.D. del. R.M. ubern. luth.

Monteru Bros. imp.

TUMOUR IN THE BLADDER REMOVED BY PERINEAL INCISION.

COMPLETE RECOVERY.

BY

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(Received April 8th—Read June 3rd, 1879.)

W. N—, æt. 21, light complexioned, healthy looking, was admitted into Addenbrooke's Hospital, Cambridge, on September 17th, 1877. Six weeks previously he began to feel pain at the root of the penis after micturition, and the desire to pass water became frequent. At the time of his admission there was, in addition, blood in the urine after any exertion; but this subsided completely, or nearly so, when he remained in bed. He several times made the attempt to get up; on his doing so, however, the blood invariably reappeared, and the other symptoms were increased. Occasionally the flow of water suddenly stopped during micturition. There were pus and blood-corpuscles in the urine, also crystals of oxalate of lime, and epithelial scales, but no casts from the kidneys.

I sounded him on two or three occasions, but could not discover a calculus or obtain any other information as to the nature of the disease. The sounding was always followed by bleeding. Under treatment the oxalate crystals disappeared, but no improvement in the other symptoms took place. On

the contrary, they became more severe, especially the pain and straining which were relieved only by opium. A flexible catheter was left in the bladder, but it could not be borne. A firmish mass could be felt above and behind the pubes and from the rectum; it appeared to occupy the position of, and be connected with, the bladder. The patient wasted, and his sufferings were so great that I determined to make an incision into the bladder, for the purpose of ascertaining the precise nature and situation of the disease, and of taking any further steps which might offer a prospect of relief. If the disease were merely inflammatory and ulcerative, a free exit for the contents of the bladder might prove beneficial. If, as there was much reason to judge, a growth had taken place into the bladder, the operation would do no harm, and there might be a possibility of removing the growth.

Accordingly, on October 17th, I cut into the bladder on a staff introduced through the urethra, making the usual incision for the lateral operation of lithotomy, and, introducing my finger, found the bladder occupied by a firm mass, about the size of an orange, with a ragged surface. It was attached by a pedicle, as thick as my finger, to the interior of the bladder near the orifice of the right ureter. Partly with the finger and partly with forceps, I contrived to tear through this pedicle, and then extracted the detached mass with lithotomy forceps. I next, with my finger-nail, scratched out what I judged to be the root of the polypus, taking care not to perforate the coats of the bladder, for fear of admitting the urine into the cellular tissue of the pelvis. The growth was of moderately firm structure of the kind called fibroma or fibro-sarcoma.

During four and twenty hours after the operation, great pain was experienced about the region of the bladder; the urine flowed through the wound. For two days subsequently there was comparative ease. Then the pain returned with even greater severity than before the operation. It was relieved only by subcutaneous injections of morphia, the quantity of which we were obliged to increase till it amounted to three grains in the twenty-four hours. In

the intervals, when the influence of the morphia was passing off, his cries were loud and incessant. The wound became coated with phosphates and the bowels were very constipated, evacuations being obtained with much difficulty through the agency of medicines and enemata. This state of things went on for about two months. We supposed the disease had returned or had been incompletely removed, and we had little hope of his recovery. After that time, however, to our surprise, he began to mend; the pain diminished, and the quantity of morphia was lessened; the wound assumed a healthy appearance and healed up; the urine was passed by the natural passage without pain and at longer intervals. In the early part of January, 1878, he was well enough to return home, and was quite well and at work in January last.

A case similar to the above is related¹ to have occurred in the practice of Professor Billroth. The patient was a boy, æt. 12, with frequent painful micturition. A tumour could be felt in the region of the bladder, and it was inferred, from an examination with the sound, that it was connected with the back of the bladder. The lateral incision into the bladder was made, and a tumour, nearly of the size of a fist with an uneven surface, was found projecting from the posterior wall into the cavity of the bladder. Owing to its size it was found impossible to extract the tumour with the finger through the perineum. A suprapubic incision was then made, both recti were cut across, and a transverse incision carried into the bladder. The tumour was then torn through, near its base, with the finger, and the pedicle dissected out. It appeared to take its origin from the muscular coat, and had not attacked the peritoneum. A drainage tube was passed into the bladder and drawn out at the incision in the perineum. In a month the patient was discharged perfectly well. The tumour was principally a myo-sarcoma, and in some places myo-carcinoma.

These are, I believe, the only two cases in which the male bladder has been cut into with success, for the purpose of removing a growth in the interior. Desault is said to have

¹ 'Brit and For. Med.-Chir. Rev.,' 1876, p. 226.

twisted off a fungus from the bladder of a male on whom he was performing the operation of lithotomy. In a man, æt. 49, operated on by Gersung, the growth was not reached, and the patient died.¹ In a male child, whose symptoms gave suspicions of calculus, Crosse cut into the bladder; vascular grape-like bodies projected into the wound and were cut off. He endeavoured to remove them more effectually, but they were parts of a large broad-based growth. The specimen represented in plate xx of his treatise on 'Urinary Calculus,' is in the Museum of the Royal College of Surgeons.

Growths have been removed through the female urethra, after dilatation or cutting, with success, by Warner,² Alexander,³ Lawson Tait,⁴ and Bryant.⁵ Norton⁶ removed one by an incision through the vagina, but the patient died.

Having recently taken some pains to investigate the subject of growths in the bladder, I subjoin a brief account of them.

They may occur at any part of the male or female bladder, but are most frequent near the orifices of the ureters.

They may present the following characters:

First. They may be *VILLOUS* or *PAPILLOMATOUS*, arising from the surface of the mucous membrane, and consisting of more or less dilated blood-vessels covered by polygonal variously shaped epithelial cells. They are at first, and may long remain, solitary; but similar secondary growths often occur near by or at a distance, and the mucous membrane may acquire a coarsely granular character in the neighbourhood. The primary growth has commonly a narrow base, and, sprouting into the bladder, becomes polypose. The secondary growths may be polypose or sessile, those which are of longest standing being most polypose.

They are not very uncommon. They occur most frequently after puberty. Indeed, I do not know of any instance in which

¹ Quoted in a paper by Dr. Hudson, in 'Dublin Journal of Medical Science,' June, 1879.

² 'Cases in Surgery,' 1761, p. 264.

³ 'Lancet,' 1879, vol. i, p. 84.

⁴ 'Diseases of Women,' p. 81.

⁵ 'Brit. Med. Journal,' May 31st, 1879, p. 821.

⁶ *Ibid.*

the disease has occurred before puberty; and they may always be removed without interfering with the muscular coat of the bladder.

They do not always occasion any symptoms or inconvenience; and it is probable that they may exist for years without doing so. In a case I lately attended there had been occasionally recurring severe and prolonged attacks of hæmorrhage over a period of seven or eight years. In the intervals, which were of several months or a year duration, the urine was quite free from blood, and the patient suffered but little. He died after a period of excessive hæmorrhage. There was a pedunculated villous growth of the size of a walnut, near the orifice of the right ureter, a smaller growth of the same nature, sessile, at a short distance from it, and a granular state of the adjacent mucous membrane. In the Museum of St. George's Hospital is a specimen (ser. xii, No. 113) of a solitary villous growth, attached by a narrow base to the neck of the bladder, from a gentleman aged 81. The first attack of hæmaturia, lasting eight months, was twenty years before death. No return for four years. After which returns at intervals; the blood, which was always bright, increased to a certain point, then diminished but did not disappear entirely. Small fleshy growths in the eye of the catheter shortly before death disclosed the nature of the disease. In another specimen, 113a in the same museum, where the base of the growth is larger, an attack of hæmaturia occurred nine years before the death of the man, which took place at the age of sixty. Apparently there was no return and no symptoms till two months before death; it was then constant, with pus, &c. In the account of a specimen (2006) in the College of Surgeons, a quantity of blood is said to have been passed sixteen years before death; this subsided, and no return took place till six or seven years before death; then more severe hæmaturia, which subsided, recurring after three years; it again subsided to recur two years before death. In the last two years the attacks were more frequent, but in the intervals the patient was perfectly well. Brodie¹ remarks that he has known the

¹ Urinary Organs, p. 121.

disease protracted for seven or eight years, and the urine to be bloody for a short time, then become clear and continue so for one or two years, when the blood has again shown itself, never wholly disappearing afterwards. I do not think this point of the occasional cessation of the symptoms for long periods, which has an important bearing on diagnosis and treatment, has been sufficiently recognised.

Secondly. The growths may have their origin in the deeper layers of the mucous membrane or in the submucous tissue. These may be called **MUCOUS GROWTHS** or **MYXOMATA**, inasmuch as they resemble the mucous polypus of the nose, being composed of loose connective or filamentous tissue, with an epithelial covering which gives them a smooth surface. This covering they may lose and may become ragged, velvety, or ulcerated. They may be pendulous, hanging by one or many stalks, or may form lobulated or cauliflower-like masses. They may spring from one spot, but commonly involve a considerable surface of the bladder. They do not encroach upon the muscular coat. They are most common before puberty. Specimens in Guy's Museum 2104¹ 28, 30, 34; in St. Bartholomew's 27 and 29; in College of Surgeons 1999 and 2000 (Crosse's case), appear to be of this nature.

Thirdly. The **FIBROUS GROWTHS** or **FIBROMATA**. These are of firmer, more fibrous, structure. They originate in the submucous tissue, and are usually pedunculated. They carry the mucous membrane before them, and are often filamentous or ulcerated. Of this nature was the growth in my patient, also apparently in the case of Billroth; and in specimens 12, 44, in the Museum of the College of Surgeons, 2104¹ in Guy's, and 2000 in the College of Surgeons appear to be of this nature.

Fourthly. The growths may begin in the muscular tissue, become polypoid, and involve all the surrounding tissues.

Of these



polypose, and limited to the submucous tissue, are the most favorable for operation.

The papillomata, which are more frequently met with than the fibromata, may affect a larger area of the surface, and may be numerous. Still, as they are confined to the surface of the mucous membrane and are often polypose, they, for the most part, admit of being removed. When they are solitary this could be done without much difficulty. In none of the specimens of myxoma that I have seen would the attempt at removal have been likely to succeed. In the malignant or cancerous diseases the attempt should scarcely be made.

The diagnosis of these growths from one another, and indeed from other affections of the bladder, I need scarcely say is often difficult or impossible without digital exploration. That method of investigation will, I doubt not, in the future be more freely resorted to. The success which, in many cases of chronic inflammatory affections of the female bladder, has resulted from dilatation of the urethra and introduction of the finger, justifies us in looking with favour upon the resort to a similar method in the male in cases of protracted or serious and doubtful disease of the bladder. The requisite incision upon a staff is a matter of little difficulty or danger. The effect of it, if the affection be inflammatory, may not unlikely be beneficial. If the affection be cancerous or irremediable no great harm will have been done. If a fibrous or papillomatous growth be discovered, it may admit of being removed through the wound as in my case (or through the urethra as in Warner's and other cases in the female). Billroth's case shows that, if the growth cannot be removed, the operator should be prepared to open the bladder above, or rather behind the os pubis, for which the perineal incision, or the dilatation of the urethra, will have been a good preliminary proceeding.

THE MOVEMENTS OF THE EYELIDS.

BY

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THE movement of the eyelids, and the mechanism by which it is effected, have received very little systematic attention. In the following attempt to explain them it has been necessary to state some familiar facts in order to describe clearly other facts which are not commonly recognised.

The normal position of the lids, when separated, is such, in most persons, as to leave the cornea and iris unconcealed. The extent to which the globe is exposed, varies, however, according to the size of the palpebral fissure, to the prominence of the eyeball, and the position of the upper lid. The same degree of exposure is approximately maintained in the upward and downward movements of the eyeballs, except in the extreme degree of each. Thus, the lids are lowered and raised with the eyeball. The importance of this movement for vision is obvious. The upper lid can also be raised or lowered voluntarily in a considerable range of movement, and the lower lid in a much smaller extent of movement.

The two lids can be approximated, and by a greater degree of this movement they can be pressed firmly together. What is the mechanism by which these several movements are effected?

For their performance two muscles only exist: the levator palpebræ superioris, which raises the upper lid, and maintains it in elevation, and the orbicularis palpebrarum, which can lower the upper lid and raise the lower, and so bring the two together—gently when the palpebral portion contracts, forcibly when the outer circumferential part is called into action. The unstriated fibres of Müller have probably no influence on the voluntary movements of the lids.

It will be at once evident, that these two muscles, the levator and the orbicularis, do not account for all the movements of the lids, for there is no muscle which can effect the depression of the lower lid. It is probable, indeed, that these muscles have an even smaller share in the production of some of the movements than might, at first, be supposed.

Another mechanism which may be concerned in some movements of the eyelids is the movement of the eyeball. What connection exists between the two? The only direct connection is the reflection of the conjunctiva from one to the other, but the membrane at its reflection is so loose that little, if any, influence can be exerted by this connection, as experiment on the dead body readily shows. A more important relation is that of position. The surface of the eyeball presents two curves, one of the cornea, the other of the sclerotic. At the junction of the two is a depression which may be termed the sclero-corneal sulcus, and in this, in most persons, the edges of the eyelids rest when they are separated, and the expansion of the tarsal "cartilage" lies against the convexity of the sclerotic, and is kept in contact with it by the elasticity of the cartilages and by the tonic force of the orbicularis. The equatorial region of the surface of the globe is distant from its centre about one millimeter more than the bottom of the sclero-corneal sulcus. The pressure of this convexity against the tarsal "cartilage," when the globe is moved, appears to be a mechanism capable of moving the upper lid down, and

the lower lid up, with the eyeball. The pressure of the corneal prominence against the edges of the lids, may also move them with the globe, and it is probable that the lower lid is thus depressed, but the prominence of the cornea appears insufficient to affect the position of the upper lid.

The influence of the elasticity of the tarsal "cartilages," and of the tone of the orbicularis, in maintaining the eyelids in contact with the globe, is seen in the fact that the lids are to a considerable extent moulded on the eyeball, so that the point of widest separation of the lids corresponds to the middle of the cornea, and if the position of the latter is changed, as, for instance, in long standing paralysis of the external rectus, the inner part of the palpebral fissure becomes the widest part. Indeed, a similar, although slight, change in the shape of the fissure may in some individuals be observed to accompany the lateral movements of the eye in the normal condition.

The movements of the lower eyelid may be conveniently considered first, because they constitute a more simple problem, on account of the absence of any other muscle than the orbicularis. When the lids are gently closed the lower lid moves upwards in consequence of the contraction of the orbicular fibres, which, having their point of attachment near the inner canthus, may be seen to move the skin towards their attachment in raising the eyelid. This movement is slight, not more than two millimeters, no doubt because the point of attachment of the fibres is nearly on a level with the edge of the lower lid. In forcible closure of the lids, the orbicularis acts more strongly, and the extra-palpebral portion pushes up the tissues outside the lid, and so increases the degree and force of its elevation. When the eyelids are separated, the orbicularis relaxes, and the lower lid descends to its former position, in consequence apparently of its elasticity, aided perhaps by the prominence of the cornea. It is possible that its descent may be aided by the tonic force of the fibres of Müller.

The lower lid follows the movements of the eyeball upwards and downwards, but not very closely. The globe gains

upon the lid in each movement, so that in the extreme downward movement the edge of the lid covers the lower edge of the cornea, and in the extreme upward movement about two millimeters of the sclerotic are exposed below the edge of the cornea. No muscular mechanism exists which can cause the downward movement. It must, therefore, be the effect of the movement of the eyeball acting upon the lid. The action is not by means of the reflection of the conjunctiva, for this is so loose that the lid can be raised passively so as to cover the lower half of the cornea, and it cannot explain the descent of the lid, which commences as soon as the globe begins its downward movement. The depression of the lid must, therefore, be the result of the pressure of the cornea against the edge of the eyelid. This force will be uniform, or nearly so, in its action, and since the resistance to the movement—the elasticity of the attachments of the tarsal “cartilage”—will increase with the movement, the effect upon the lid progressively diminishes as the movement proceeds. Hence the globe gains upon the lid. When the eyeball returns to its normal position, the elasticity of the attachments of the lid brings it back to its place of rest with the edge in or near the sclero-corneal sulcus.

What is the cause of the upward movement of the lower lid when the eyeball is rotated upwards? The only muscular mechanism which can cause this movement is the contraction of the lower portion of the orbicularis. But if the eyelid is watched during the movement no contraction of the orbicularis can be observed. On the contrary, instead of any wrinkling or movement of the skin, such as always results from, and indicates, the contraction of these fibres, the tissues of the lower lid are extended much more than when the eye is in the mid-vertical position. Moreover, in paralysis of the orbicularis from disease of the facial nerve, the movement upwards of the lid with the globe is the same as under normal conditions. The movement must, therefore, be ascribed to the pressure of the lower convexity of the sclerotic against the tarsal “cartilage”—pressure which can be readily seen or felt if the finger is placed upon the lower lid during the

movement. The amount of elevation varies in different individuals, and is related, in part at least, to the prominence of the eyeball. If this is greater or less than normal the movement is less ; because if greater, the eyelid is already stretched over the convexity of the globe to a degree which renders the movement of the eyeball incapable of moving the lid to any considerable extent ; and if less, the sclerotic does not come forward sufficiently to press against the lid ; the relation on which the movement depends in the former case being already in operation, and in the latter not occurring at all.

Movements of the upper eyelid.—The upper lid possesses a greater range of movement, and a more complex mechanism, a special muscle being necessary, partly on account of the range and importance of its movement, and partly on account of the degree to which the lid is under the influence of gravitation, by which its descent is facilitated.

It is maintained in the normal position of elevation by the tonic action of two muscles, the levator and the orbicularis, that of the former preponderating. The influence of each of these muscles can be recognised when the other is paralysed. When the levator is paralysed, there is the familiar ptosis ; when the orbicularis is paralysed, it may be observed that the upper lid is a little higher on the paralysed than on the unparalysed side ; higher, that is, in the degree to which the levator is unopposed by the tone of the orbicularis.

The ptosis in paralysis of the levator demonstrates that the support given to the edge of the lid by the prominence of the cornea is insufficient to prevent the descent of the lid under the influence of the orbicularis and of gravitation.

When the lids are gently closed by a voluntary effort, the upper lid is lowered by a gentle contraction of the orbicularis, demonstrated by the fact that the lid descends further than it does in complete paralysis of the levator, and by the fact that in complete paralysis of the orbicularis this full descent of the lid does not occur. The closure of the lids during sleep is also effected by a gentle tonic contraction of the orbicularis. But for this movement to occur the

levator must be relaxed, and in consequence of this relaxation, when the orbicularis is paralysed and an attempt is made to close the eyes, the upper lid falls.

When the eye is "opened," the upper lid is raised by the contraction of the levator; with this is often associated a synergic contraction of the frontalis. In some persons this occurs even on gentle elevation, and in all persons in extreme elevation.

The upper lid follows the movements of the eyeball much more closely than does the lower lid, the edge being commonly maintained in juxtaposition to the edge of the cornea. The descent of the lid, on downward rotation of the globe, is not due to the contraction of the orbicularis (which causes its descent in voluntary closure of the eyes). This is proved by two facts. (1) The movement is unattended by any wrinkling of the skin such as can be observed in even slight contraction of the orbicularis. (2) The lid follows the downward movement of the globe almost, sometimes quite, as accurately if the orbicularis is paralysed as in the normal state. Gravitation no doubt assists the movement, but it is probable that the chief mechanism is the movement of the eyeball itself, the pressure of the upper convexity of the globe against the tarsal "cartilage." If the movement is closely watched, it is difficult to resist the conviction that this is the chief agency, since the upper surface of the eyeball can be seen to be rolled against the eyelid and to bulge it forwards. That this agency is effective, in addition to gravitation, is evident from the fact that when the orbicularis is paralysed, and an attempt is made to close the eyes (*i.e.* when the levator is completely relaxed and the lid is left to the influence of gravitation), it does not sink so low as it does when the eyeball is rolled downwards. The influence of the movement of the globe on the lid is increased by the circumstance that the attachment of the tarsal cartilages is considerably below the middle of the globe.

For this descent of the upper lid, in the downward movement of the eyeball, there must be a relaxation of the levator. This may appear self-evident, but it is necessary, on account

of certain pathological facts, to be presently described, to consider the proof of this relaxation.

(1) The extent of movement is so great that it is scarcely possible that it can be produced by the cause just assigned against the tonic contraction of the levator. (2) The eyeball and eyelid move uniformly; the force moving the lid acts uniformly; if the movement of the lid were opposed by so strong an elastic force as the unrelaxed tone of the levator, there would be a progressive deficiency in the movement of the lid compared with that of the globe. The uniformity in the movement, therefore, implies a relaxation of the levator in exact proportion to the movement of the globe. (3) The levator and frontalis have been seen to be associated in action, and when there is a considerable permanent tonic contraction of the frontalis (as normally in some individuals, and in partial ptosis), the eyebrow may be observed to descend, in consequence of the relaxation of the frontalis, in exact proportion as the eyeball is moved downwards and the upper lid descends. These considerations prove that there must be an accurately proportioned relaxation of the levator permitting the descent of the lid.

When the eyeball, having been rotated downwards, is again raised, the upper lid is raised with it. This is not by the influence of the globe on the lid, for, as has been already shown, the phenomena of ptosis demonstrate that the prominence of the cornea is insufficient to support, and, *a fortiori*, to move the lid. The elevation is by a contraction of the levator, corresponding in degree to the contraction of the superior rectus, just as the levator was relaxed in exact proportion to the contraction of the inferior rectus. The occurrence of this contraction of the levator, in upward movement of the eye, can be readily observed in the following manner. If the eyeball, after having been directed downwards, is quickly raised, the elevation of the lid by the levator is in excess of the movement of the globe; it is raised too high, and subsequently falls to its proper position.

This action of the levator, associated with that of the superior rectus, is entirely beyond voluntary control. In the

simulated ptosis of hysteria, for example, the upper lid is kept down by a gentle contraction of the orbicularis. But if the patient is made to look up, the levator contracts synergically with the superior rectus, and a strong contraction of the orbicularis is necessary to prevent the lid being raised.

The association of the levator and superior rectus suggests that both are relaxed in similar degree when the eyeball is moved downwards, in accordance with the law which, it is evident, must obtain in all muscular actions, that the opponents of the muscles producing the movement are relaxed in exact proportion to the degree of movement.

In gentle closure of the eyelids the levator is also relaxed, but the eyeball remains stationary,¹ the relaxation being associated with contraction of the orbicularis, the state of the recti being unaltered. In strong contraction of the orbicularis, however, the eyeball is, as is well known, rolled upwards. For this there must be contraction of the superior rectus, the inferior rectus being presumably relaxed. The levator is relaxed in forcible, just as in gentle, closure of the lids. This is proved by the circumstance that in facial paralysis the lid does not rise during the strongest attempt to close the lids.

It may be well to group these associations in a tabular form.

		Looking down.		Closing eyelids.		
				Gently.	Forcibly.	
Orbicularis	. .	No action	...	Contraction	...	Contraction
Levator	. .	Relaxation	...	Relaxation	...	Relaxation
Superior rectus.	. .	Relaxation	...	Inaction	...	Contraction
Inferior rectus .	. .	Contraction	...	Inaction	...	Relaxation.

Thus in the upward and downward movements, the state of the levator corresponds to that of the superior rectus, and is the opposite of that of the inferior rectus, and in the forcible closure of the lids, the state of the levator is the opposite of that of the superior rectus, and the same as that of the inferior rectus.

¹ This is commonly the case during sleep, natural or artificial. The statement that the eyeballs are rolled upwards during sleep, which has been current since the time of Sir Charles Bell, is, according to my own observation, true only of a small minority of cases.

From this it seems probable that the centre for the forcible closure of the lids is physiologically distinct from that for their gentle closure.

The association of the superior rectus and levator limits very much the voluntary action of the latter. The state of the superior rectus, in any position, represents nearly the maximum voluntary contraction possible of the levator. This muscle may be completely relaxed, and again contracted, but only to the degree in which the superior rectus is contracted. Hence, in each position, the lid cannot be raised much (in some persons not at all) above the edge of the cornea. If the eyes are directed downwards, for instance, the eyelid is in the same position as when the lids are closed and the eyes directed forwards. In the latter case the lids can be raised readily to the full extent, in the former case scarcely at all. This is the result of the central mechanism, for there is no local connection which prevents the movement, as experiment on the dead body readily shows.

The mechanism or path for the voluntary elevation of the lid lowered in closure of the eyelids, is, no doubt, distinct from that for the conjoint elevation of the eye and lid by the action of the levator and superior rectus. This fact, evident from the above physiological considerations, is proved also by a pathological fact presented by the patient now shown, who is in the National Hospital for the Paralysed and Epileptic, under the care of Dr. Hughlings Jackson, whom I have to thank for permission to exhibit the case. She is suffering from partial paralysis of the third nerves (probably central) causing double ptosis. When the eyes are directed straightforwards, the upper lid of each eye half covers the cornea, and she cannot by the strongest voluntary effort raise it from that position. But if she looks up, rotating upwards the eyeball, the eyelid is raised as high as normal, so as to uncover the cornea almost completely. There is thus partial paralysis of the separate action of the levator, in raising the depressed eyelid, but no paralysis of the action associated with that of the superior

rectus. This phenomenon (which I have observed in other cases) constitutes a pathological proof of the separateness of the mechanism for the different actions of the levator, which has just been inferred from physiological facts.

Other phenomena of paralysis present modifications of the movement of the eyelids, as they have been above described. One of these especially deserves mention.

If the orbicularis is paralysed, the relaxation of the levator occurs on an attempt to close the eyes, just as when the orbicularis is unparalysed. The upper lid falls, but the lids cannot be brought together. If, however, the inferior rectus is paralysed, so that the eyeball cannot be rotated downwards, an attempt at this movement is not attended by the relaxation of the levator, which, as we have seen, usually accompanies the downward movement of the globe. I show photographs of two patients presenting this condition (Plate IX). When the patient looks down, the upper lid, on the unaffected side, falls with the eyeball. On the affected side, on which the eyeball is not rotated down, the eyelid does not fall but remains in the position corresponding to that of the globe (Plate IX, fig. 2), and yet, on closing the lids, the upper one falls in the natural manner (Plate IX, fig. 3).

Thus in the absence of the downward movement of the globe, the attempt at the movement does not cause the relaxation of the levator which accompanies the actual movement. But relaxation implies inhibition of the centre which is in tonic action. The phenomena of "secondary deviation" of the eyes, would lead to the *à priori* expectation that a fruitless attempt to move the eye down would cause an increased inhibition of the levator, instead of which there occurs no inhibition at all. The practical convenience of the arrangement is more obvious than is its mechanism. The difficulty in explaining it is increased by the fact that, as just stated, the relaxation of the levator, associated with the contraction of the orbicularis, does occur when the latter is paralysed, on attempting to make it contract.

The best explanation which occurs to me is that the inhibition of the levator is not primarily the result of a central

mechanism, but is a reflex result of the effected movement. The afferent impression causing such an inhibition may originate from the contracting fibres of the inferior rectus, or from the tension on the fibres of the levator by the movement of the globe—its pressure against the tarsal cartilage being, as we have seen, the chief agent in causing the descent of the lid. It is certain that the relaxation of the levator is in exact proportion to the movement of the globe. The phenomenon is of much interest since, whatever is its mechanism is probably also the mechanism by which the relaxation of the opponents of other contracting muscles is effected.

The last point to which I would direct attention is, that the eyelids, the upper one especially, participate in the movements of the eyeball in vertical nystagmus. The mechanism for this movement is, no doubt, the same as for the other movements of the eyeball, and partly the result of the association of the levator and superior rectus, which is as marked in disease as in health.

NOTE.—Sir Charles Bell, in his work on the Nervous System, expressed the opinion that the contraction of the levator palpebræ superioris, at the same time that it raises the upper lid, presses forward the eyeball, and that this movement of the globe pushes down the lower eyelid. The assumption is, however, very difficult to prove on account of several circumstances which Sir Charles Bell did not take into consideration. Certainly the chief part of the descent of the lower lid which occurs when the upper lid is raised is due simply to the relaxation of the orbicularis, which, even in the gentlest closure of the lids, raises the lower lid to meet the upper, and, relaxing, allows it to fall when the upper lid is raised. If the eyeballs are kept directed downwards when the eyelids are opened, the levator probably does not act at all, but the lower lid falls as much as, or more than it does during the strongest action of the levator, and the movement of the skin shows that this depression is due to the relaxation of the orbicularis. Sir Charles Bell's opinion was probably

founded on the fact that if the finger be placed against the lower eyelid while the upper lid is being raised the lower part of the globe is felt to be pushed against the finger. But it is doubtful whether this is to any extent the result of the contraction of the levator. The degree to which the eyeball can be felt to be pushed against the lower lid depends on the degree of rotation upwards of the globe with which the action of the levator is associated, and it is mainly due to the rotation of the eyeball causing the lower prominence of the sclerotic to project. Moreover, the relaxation of the orbicularis relieves the eyeball from the slight backward pressure, and consequently a slight movement forwards may result from this cause. Having regard to the action of these two causes, it seems uncertain whether the levator protrudes the globe, as Sir Charles Bell supposed.

DESCRIPTION OF PLATE IX.

(The Movement of the Eyelids. W. R. Gowers, M.D.)

Figs. 1—3.—Case 1.—Partial paralysis of left third nerve, showing that the upper eyelid remains elevated if the eyeball does not move downwards in looking down.

Fig. 1.—Position of lids on looking directly forwards.

2.—Looking down: the right eyeball is rotated downwards, the left not (paralysis of left inferior rectus); the left upper eyelid remains raised, the levator not being relaxed.

3.—Gentle closure of eyes, complete relaxation of levator on contraction of palpebral part of orbicularis.

Figs. 4—6.—Case 2.—Paralysis of left inferior rectus, showing the same fact.

Fig. 4.—Position of lids on looking directly forwards.

5.—Position of lids on looking down: the right eye is rotated downwards and the right lid falls; the left eyeball and lid remain almost in the same position as in the first figure.

6.—Gentle closure of eyelids; complete relaxation of levator and descent of lid on gentle contraction of the palpebral part of the orbicularis.



Fig. 1.



Fig. 2.



Fig. 3.



Fig. 4.



Fig. 5.



Fig. 6.

OBSERVATIONS
ON THE
OPHTHALMOSCOPIC APPEARANCES
IN THE
TUBERCULAR MENINGITIS OF CHILDREN.

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THE following remarks are based upon the ophthalmoscopic examination of 26 cases of tubercular meningitis, out of a total of 31 that were admitted into the Hospital for Sick Children whilst I was registrar there. I am indebted to the staff of the in-patient department, Dr. Dickinson, Dr. Gee, Dr. Cheadle, and Mr. Smith, for permission to publish these cases.

Out of the 31 cases, 5 have been excluded for the following reasons:—In 3, no examination had been made of the optic disks; in one only one disk; in the remaining case tubercular meningitis supervened in a case that was under observation for a cerebral tumour. Of the remaining 26, a table is appended, showing the optic changes and the coincident symptoms, with the post-mortem appearances of the brain and its membranes in each case.

In cases of tubercular meningitis recognisable changes

occur frequently in the fundus oculi, perhaps in at least 80 per cent. In many they aid materially the recognition of the disease, whilst in a few they enable a pretty certain opinion to be given when the other symptoms only suggest its existence; in most they only supplement the other evidence.

The changes I have found may be thus summarised :

1st. Hyperæmia of the disk, usually transitory, more distinct early on the inner side; some veiling of the margin, partly due to similarity in colouring of the disk and choroid; some disproportion between the veins and arteries almost as $1\frac{1}{2}$ to 1; with darkening of the veins.

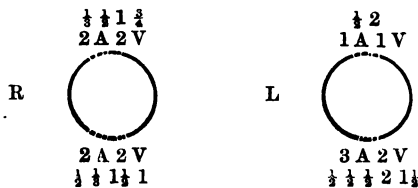
2nd. Lessening of the redness; loss of margin, seen first where the vessels cross the margin, then inside, and finally outside; a degree of swelling, always moderate, affecting especially the margins of the disks and adjacent retina; greater disproportion between the veins and arteries, the proportion being about as 2 to 1; the arteries small and pale, the veins dilated and bent; as the swelling increases often a white shining line appears on each side of, and parallel to, the vessels on the disk, especially the arteries; with obscuration of parts or of the whole of a vessel, especially at the margin of the disk.

But in another set of cases the appearances present altogether a different character, viz. marked changes in the retinal vessels, with or without a little reddening of the disk, but unaccompanied by swelling or obliteration of the margin; there is, in fact, a marked disproportion between the vessel and disk changes in this class.

In order to give full value to these alterations in the fundus, we must know whether the normal disk does not present variations in its appearance, simulating the pathological changes; this is unfortunately the case, and to such an extent that in many cases the progressive change in the disks, that is, the variation from the condition found on one day to that present on the next, gives better evidence than will a single examination in another case, although this may disclose a greater degree of change.

The examination by the direct method of the disks of healthy children confirmed what has previously been observed as to their variability in different cases. In few is there absolute similarity between the disks of opposite sides as to the position and amount of a well-defined margin. It is rare to find a perfectly clear margin all round, some obscuration almost invariably affecting the part where the vessels cross, and of the remainder the inner margin is far more frequently affected than the outer. Variations in the colour of the disk occur, and the inner half is frequently reddened. The arteries and veins vary in size from an equality to a proportional size of about 1 to $1\frac{1}{2}$, and either may be partially hidden in their passage over the disk; the veins are darker in some disks than in others. The apparent depth and distinctness of the physiological cup varies markedly.

A consideration of these possible variations in health makes it evident that, unless the normal condition of the eye has been previously noted, a single examination will in many cases teach us little; we must trust to a subsequent examination, when, if we have to deal with actual changes, the chances are in favour of some alteration from the condition previously found. A concise note may be made of the case by drawing two circles to represent the disks, the clear margin being shown by a continuous line, and the blurred part in its proper position by a dotted one, the number and relative size of the arteries and veins being shown by numbers above and below the respective disks. In the absence of swelling we can only by thus tracing its onset distinguish the pathological from the physiological veiling of the margin. The following is an example of a case thus delineated :



As to the optic changes in tubercular meningitis, the following questions suggest themselves :

1st. The proportion of cases in which changes occur.

2nd. The number in which they occur sufficiently early to be an aid in diagnosis.

3rd. The condition of the brain membranes, &c., and their relation to the optic changes.

1st. As to the frequency with which changes in the fundus oculi may be expected. In the twenty-six cases tabulated below, and in all of which tubercles were found in the membranes of the brain, in only four were optic changes absent or so slight as not to be certainly pronounced as abnormal; this gives a percentage of about 84. It may be observed that this agrees pretty closely with the numbers given by Dr. Clifford Allbutt and M. Bouchut. Now, although the recognition of optic changes when well marked is quite easy, it is difficult in many cases to positively exclude their existence, unless the previous condition of the disks have been taken into account, and the examination continued up to within a short period of death. We may well consider that decided changes occur in about 80 per cent. This alone makes it a sign of great importance, and especially so when we consider that its evidence is much more definite than the vomiting, headache, pulse changes, &c., which form the early symptoms of tubercular meningitis.

2nd. The number of cases in which they occur sufficiently early to be of diagnostic value. About this point it is not so easy to obtain evidence, because the eyes are not usually examined before the symptoms are suggestive, and also the children admitted as in-patients are mostly advanced in the disease and pronounced in the symptoms; and even in those cases in which the ophthalmoscope aided or even made the diagnosis its value cannot properly be expressed by a written description of the case, unless this be made very long; for so much depends upon the aspect and mental condition, the intensity of the symptoms, &c.; the mere date before death of the appearance of the changes gives but little idea of the

actual value of the sign. But in order to make some definite standpoint I have taken the advent of palsy, or of decided and continuous unconsciousness, as the time when any ophthalmoscopic changes will be only confirmatory; where found before this they aid or even enable us to make the diagnosis.

Below is given a separate table of six cases out of the twenty-six examined, in which the ophthalmoscope was of decided service; in two of these it may be said to have made the diagnosis, viz. in Nelly J—, whose case lasted twenty-six days; here, with equivocal history and at the time indefinite symptoms, the optic disks showed, on the fourteenth day, unmistakable changes, viz. œdema and disproportion of the retinal arteries and veins, whilst it was not till the nineteenth day that the symptoms would have made the diagnosis certain. It may be noted, in confirmation of this statement, that Dr. Gee, on the fifteenth day, made the note that the diagnosis of tubercular meningitis rested on the optic changes. In the other case, William C—, the ophthalmoscopic changes were present on the ninth day, the diagnostic symptoms on the fifteenth, the child dying on the twentieth. In both these cases the meningitis was chiefly expended on the optic commissures, the membranes here being greatly thickened with lymph, whereas there was little in the membranes of other parts. This explains the appearance of the optic changes before the development of the other symptoms, and probably represents the class of case in which the ophthalmoscope will be of greatest assistance. But these 6 cases do not probably represent all in which the ophthalmoscope might have aided, for in Case No. 7 the optic changes were so pronounced on the first day of examination, when the case was from its symptoms well defined, that they would probably have been found if looked for a day or more before, when the symptoms did not indicate so clearly the nature of the disease. The same statement applies, though in a less degree, to Case 9.

Cases in which the ophthalmoscope aided the diagnosis.

Name.	Duration of case.	Date of ophthalmoscopic changes.	Symptoms coincident with and preceding the changes	Date of appearance of marked symptoms.	Remarks.
William G. Case 4	16 days	13th day	Headache and nausea; restless nights; fretful days. Abdomen flat; pulse slow, irregular	14th.—Weakening of left facial.	—
John M. Case 8	12 days	6th day	Tongue furred. Vomiting and headache. Restless nights. Bowels confined. Not looking ill. Reading a book	7th.— External strabismus. Palsy left face, arm and leg	Aspect on sixth day not cerebral.
Minnie S. Case 14	15 days	10th day	Paroxysmal cough ending in vomiting. Slight headache once. Pulse 100. Drowsy	11th.—More drowsy. Right facial weakened	—
Adolphus M. Case 17	10 days	8th day	Occasional vomiting and headache for two days. Pulse irregular. Irritability; screaming	Signs never more marked	Death from collapse of lung.
Nelly J. Case 23	26 days	14th day	Cough six weeks, often followed by vomiting. Dislike of light. Pulse regular	19th.—Screaming. Half comatose. Occasional divergent squint	Child died more from chest condition.
William C Case 26	20 days	9th day	Repeated vomiting; clean tongue; costive bowels; drowsy	15th.—Suddenly became unconscious. Pulse slow and irregular	—

There is an interesting point, which may be here mentioned, of which I have seen at least one well-marked example, viz. the diminution of the changes in the disks a few days before death. In the case referred to (No. 18) there was coincidently a marked diminution of the other cerebral symptoms, the child, after being unconscious, recovering this for at least five days preceding death; the squint and palsy diminishing, the abdomen becoming less retracted, and the child dying ultimately from the lung condition, there being, after death, barely any breathing room in either lung, from the enormous amount of tubercular deposit. The optic improvement was noticed to be progressive for two days, and would have probably continued but for the death of the child. The case is also interesting as showing a tendency towards cure.

3rd. We come to the question as to the cerebral changes with which the optic are associated, and how they are related to one another. Obstructed venous return, and extension by continuity of the inflammation from the membranes to the optic commissure and nerves, are the causes assigned for the changes observed in the fundus of the eyes; and, I believe, with some doubtful cases the ophthalmoscopic changes can be divided under these two heads, that is, we can tell whether post-mortem, inflammatory, or pressure signs will predominate. If the latter, we find, at an early stage, dilatation of the veins, with or without slight hyperæmia papillæ, whereas, in those cases in which inflammation plays a more prominent part, the vessel changes become great only when hyperæmia is marked, or probably swelling also present. Of course cases occur in which neither inflammatory nor obstructive conditions predominate, and these would give intermediate conditions of disk. The evidence of an effective intracranial pressure in those acute cases in which the cranial sutures have not time to yield is considerable; thus we have—

1st. Marked diminution in the size of the superior longitudinal sinus.

2nd. Pressure of the blood out of the veins over the convexity of the brain; firstly, over the convolutions themselves

and secondly, out of the larger veins in the sulci. (When the child dies asphyxiated these veins are often full, although there is marked flattening of the convolutions.)

3rd. The pallor of the brain substance itself.

4th. Anæmic condition of the choroid plexus in cases of large ventricular effusion.

5th. The appearance of a tortuous vein beneath the pericranium coursing along the coronal suture.

6th. Occasionally the enlargement of a cutaneous vein at the inner or outer angle of the orbit.

The active agents in producing this pressure are the ventricular and cerebro-spinal fluid thrown out in excess; these are usually in excess together, but may, I believe, be so independently; thus, in Case 9, the subarachnoid was in excess, and not the ventricular. The former gives rise, if in quantity, to a rounded and slightly translucent swelling of the sheath of the optic nerve just behind the eyeball, and which can be let out as a drop of clear fluid by snipping the coats. It would appear that this subarachnoid excess is alone capable of giving rise to dilatation of the retinal veins. Below is given a table of the cases, so as to show the duration of the cases and the character of the changes in each; from this it appears that the average duration of cases in which the disk changes precede is eighteen days; whereas in those which show only or chiefly vessel changes the cases run a course of twelve days, death occurring before the development or extension of inflammation along the optic commissure and nerves, and these latter cases appear to be characterised often by earlier development of coma and rigidity, with a less-marked tendency to unilateral palsy, for this is due to an inflammatory change being more marked and mounting higher in one Sylvian fissure than in the other. This origin of the palsy is well illustrated in Case 22, where the very first symptom of tubercular meningitis was a sudden onset of convulsions, followed by aphasia and right hemiplegia, which persisted till death, fourteen days after.

Case.	Duration. days.	Affection of retinal vessels.	Hyperæmia of disk.	Swelling. of disk.
1	15	+	+	0
2	10	+	+	0
3	10 No change.	0	0	0
4	16	+	0	+
5	16	+	+	+
6	19	+	0	+
7	12	+	0	+
8	12	+	0	+
9	21	+	0	0
10	18	+	0	+
11	9	+	0	0
12	18	+	0	+
13	22	+	0	+
14	15	+	0	+
15	20 No change.	0	0	0
16	21	+	0	0
17	10	+	+	0
18	23	+	0	+
19	4	+	+	0
20	14 No change.	0	0	0
21	12	+	0	0
22	19 No change.	0	0	0
23	26	+	0	+
24	23	+	0	+
25	12	+	0	+
26	20	+	+	+

Post mortem.—The deposit of tubercles was almost limited to the left side of the brain, and they were especially abundant about the bifurcation of the fissure of Sylvius.

It has been remarked that the ophthalmoscopic changes are most marked on the side of the greatest lesion of the brain, that is, on the opposite side to the palsy of the limbs or face. In seven cases which showed a difference between the two disks, this rule only held good in four; that is, it failed in almost half the cases; and, indeed, this is the result that might have been expected from the position and size of the optic commissure, and the equal distribution of fluid pressure. The seventh nerve is almost invariably paralysed or weakened on the same side as the arm and leg, and gives earliest evidence of its being affected by the eyelid of its side opening wider

than the opposite ; this is far more easily observable than the condition of the mouth. Sometimes the palsy shifts sides.

I regret not having had the opportunity of examining the disks in adults suffering from tubercular meningitis, as here, owing to the frequently insidious onset and rapid course of the disease, any early indication would be of special value. But in the few cases in children associated with chronic disease, viz. two of hip and two of lung, the ophthalmoscopic changes were slight in three and absent from one ; it is also noteworthy that in another case an increase of chest signs and symptoms, which consequently caused death, coincided with marked lessening of the optic changes ; hence, I am led to suspect that in many of the cases of tubercular meningitis of the adult, supervening on chronic phthisis in an insidious manner, we shall not obtain much aid from the ophthalmoscope. In two cases of rapidly fatal simple meningitis, I examined both disks in one and one in the other, with negative results ; this was due, no doubt, to the slight basal affection, and to the short duration of the cases.

As to the question of recovery from tubercular meningitis, the only possible form of demonstration is that given by Dr. Clifford Allbutt. But there have been lately in the hospital at least four cases with the symptoms more or less marked that recovered completely. I relate two, because with the symptoms in them there were well-marked and progressive optic changes, which cleared up completely but tardily after the cessation of the symptoms.

The first case, Eliza E—, æt. 8½ years, admitted under the care of Dr. Gee, gave a history of headache and wasting for six weeks ; about the middle of this time she had a slight blow on the head with a roundabout handle. A week later she had a fit, and from this time commenced to vomit. Whilst in the hospital the symptoms were marked inertness, occasional vomiting, confined bowels, a slow irregular pulse, and marked optic changes. The symptoms ceased five weeks from the fit, and a week later the disks commenced to clear up, becoming in a fortnight almost normal. The child came to be inspected eight months later, and was found to be

in excellent health and the disks were quite normal. Her sister was admitted about this time with advanced phthisis. The other case was equally marked, there was a decided phthisical history on the father's side, and the child had herself obscure signs of old mischief at one apex and was much emaciated, having wasted for the last twelve months, and complained of headache for two months. One month before admission there had been discharge from one ear for a few days, but this never recurred. The symptoms were just the same as in the other case, and the optic changes quite as marked, but in this one the improvement in the eyes and in the symptoms commenced together six weeks from the onset of vomiting, and the child was discharged well. Four and a half months later she presented herself; the disks were then found normal, and her only complaint was of occasional headache and nausea after the noise at school.

Of course it is impossible here to exclude simple meningitis, tumour of the brain, or simple optic neuritis. As bearing on the same question, Case 18 may again be referred to, which before death showed amelioration of the cerebral symptoms and lessening of the optic changes.

I have only once seen tubercles of the choroid; they occurred in a case which presented no other ophthalmoscopic changes. Dr. Gowers kindly examined the specimen microscopically and demonstrated beyond doubt their nature.

Name. Age. Remarks.	History of case before ophthalmoscopic examination.	Date of examination from onset and condition of fundus of eyes.	Symptoms present when the optic disks were first examined, and subsequent course of the disease.	Post mortem.	Duration.
Case 1. James M., 3½ years. Dr. Gee. Palsy the same side as the most affected disk	Measles six months before; not well since; wasting. 1st day.—Head- ache; drowsiness. Since then restless nights; headache; screaming out; Bowels confined. The mother had hemoptysis eight- teen months before A cough for the last six months. In- patient three weeks with debility, otor- rhea, and slight chest signs. One morning sick and drowsy	8th day.—Left disk na- tural; right disk red; 9th.—Right disk red; margin obscured, except externally. Veins large, dark, and about twice the size of the arteries. 14th.—Right, more ob- scuration of margin. Vessels as before. Left, slight reddening	8th day.—Sick once. Roll- ing the head. 9th.—Very drowsy and irritable. Pulse 96, irregular. Right facial weakened. Pulsation in neck. 11th.—Pulse 128, regular. Screaming. Head retracted. 14th.—More un- conscious. Flushed. Pulse regular. 15th.—Palsy of right arm; convulsed; death	Convolutions not flattened or sticky. No great excess of fluid in ventricles or anterior subarachnoid space. Little inflammatory thickening of the pia mater; more tuber- cles about the left Sylvian fissure than the right. Pia mater about the optic nerves inflamed. The nerves in- jected. Tubercles also in both lungs	15 days.
Case 2. Annie B., 9 years. Dr. Gee. Vessel changes and hyper- semia. Not much lymph in meninges. Little uni- lateral affec- tion	2nd day.—Left veins somewhat enlarged and arteries small; pale colour. A faint mar- gin outside, else lost. 5th.—Left disk red- dened. Vessel changes rather greater. Right vessels and disk in same condition as left; veins twice the size of the arteries. 9th.—Mar- gins quite lost in both. Disks paler. Vessels as before, not obscured in their course	2nd day.—Headache and vomiting only. Pulse 96, regular. 4th.—Unconscious. Pulse 88, irregular. Fre- quent vomiting. 9th.— Conscious; sat up. Tache marked. Bowels con- fined. 10th.—Unconscious. Convulsed right half of face. Death	2nd day.—Headache and vomiting only. Pulse 96, regular. 4th.—Unconscious. Pulse 88, irregular. Fre- quent vomiting. 9th.— Conscious; sat up. Tache marked. Bowels con- fined. 10th.—Unconscious. Convulsed right half of face. Death	Cerebral sinuses and veins of pia mater congested. Con- volutions sticky. Much fluid in lateral ventricle and subarachnoid space. Many tubercles scattered about; less lymph. Many vascular points on section of the optic nerve. Tubercular masses, size of a pea, in each lobe of cerebellum, and in left corpus striatum. Tuber- cles also in lungs, liver, spleen, kidneys, intestines, and peritoneum	10 days.
Case 3. Rose B.,	A sickly child with cleft palate. Ill	7th day.—Right natural. 9th.—Right natural.	7th day.—Persistent vomit- ing. 8th.—Pulse 148, regu-	Cerebral sinuses and veins of pia mater natural. Coronal	10 days.

6-12 years.	10-16 years.	17-20 years.	21-30 years.	31-40 years.	41-50 years.	51-60 years.	61-70 years.	71-80 years.	81-90 years.	91-100 years.
Dr. Dickin-son. No optic changes. Little inflam-mation and no pressure	first diarrhoea, vomiting, and fever, followed by consti-pation. Hurried respiration and few signs in chest	Left slight reddening	lar; respiration irregular. Drowsy. Wasting. Head retracted. Imperfect closure of left eye. 10th.—Less conscious for twenty-four hours before death. No convulsions. Temperature a little raised all along. Very obscure cerebral symptoms. Death	18th day.—Pulse 76, very irregular. Pretful and drowsy. Abdomen flat; tongue clean. 14th.—Curled up on left side; left facial nerve weakened. 15th.—Pulse 96, more regular. Unconscious in the evening. 16th.—Eyes and head turned to left; convulsed. Death	18th day.—Left disk pale. Margin obscure. Veins large, twice the size of the arteries, which are small. 15th.—Right same condition, disk and vessels	16 days.	suture not congested. Con-volutions not flattened, slightly sticky. Slight ex-cess of fluid in lateral ventri-cles. Pia mater over optic commissure opaque; many scattered tubercles; little inflammatory change. Tu-bercles also in lungs and liver	16 days.	Coronal suture marked exter-nally by a red line. Little blood in superior longi-tudinal sinus. Convolutions flattened and sticky. Much fluid in lateral ventricles and subarachnoid space. Choroid plexus and sub-stance of brain rather pale. Pia mater over optic com-missure natural. Many tu-bercles scattered about; little lymph. Caseous bron-chial glands. Tubercles in lungs, liver, spleen, and kid-neys	16 days.
Case 4. William G., 3 years. Dr. Dickin-son. Left side of brain most affected, and right disk	Fits ten months previously, and again thirteen days ago. Since the latter attack fretful days, restless nights, headache, and nausea. No constipation	13th day.—Left disk pale. Margin obscure. Veins large, twice the size of the arteries, which are small. 15th.—Right same condition, disk and vessels	18th day.—Pulse 76, very irregular. Pretful and drowsy. Abdomen flat; tongue clean. 14th.—Curled up on left side; left facial nerve weakened. 15th.—Pulse 96, more regular. Unconscious in the evening. 16th.—Eyes and head turned to left; convulsed. Death	18th day.—Left disk pale. Margin obscure. Veins large, twice the size of the arteries, which are small. 15th.—Right same condition, disk and vessels	13th day.—Left disk pale. Margin obscure. Veins large, twice the size of the arteries, which are small. 15th.—Right same condition, disk and vessels	16 days.	suture not congested. Con-volutions not flattened, slightly sticky. Slight ex-cess of fluid in lateral ventri-cles. Pia mater over optic commissure opaque; many scattered tubercles; little inflammatory change. Tu-bercles also in lungs and liver	16 days.	Coronal suture marked exter-nally by a red line. Little blood in superior longi-tudinal sinus. Convolutions flattened and sticky. Much fluid in lateral ventricles and subarachnoid space. Choroid plexus and sub-stance of brain rather pale. Pia mater over optic com-missure natural. Many tu-bercles scattered about; little lymph. Caseous bron-chial glands. Tubercles in lungs, liver, spleen, and kid-neys	16 days.
Case 5. Alice H., 5 years. Dr. Gee. Marked rigidity. Evidence of great fluid pressure	Measles four months ago, followed by otorrhoea and a cough, the latter ceasing four weeks ago. Since then complaining of headache towards evening, and rest-less at night. Marked onset seven days ago, with vom-iting, frontal headache, and drowsiness. Bowels confined. Twitch-ing in sleep	7th day.—Right disk red-dened; margin ob-scured, except exter-nally. Veins very large and arteries small, por-tion of three to one; slight swelling. Left much the same as right. 9th.—Disks not so red. 14th.—Disks redder; same changes	7th day.—Drowsy and irri-table. Pulse 98, irregular. Head retracted. Left facial weakened. Furred tongue. 9th.—Screaming last even-ing. Left arm rigid. 13th.—Pulse 100, subregular; respiration slow and deep. Unconscious. Arm and leg, left side, rigid. 15th.—Pulse rapid and small; temp. 101°. 16th.—Con-vulsions. Death	7th day.—Right disk red-dened; margin ob-scured, except exter-nally. Veins very large and arteries small, por-tion of three to one; slight swelling. Left much the same as right. 9th.—Disks not so red. 14th.—Disks redder; same changes	7th day.—Drowsy and irri-table. Pulse 98, irregular. Head retracted. Left facial weakened. Furred tongue. 9th.—Screaming last even-ing. Left arm rigid. 13th.—Pulse 100, subregular; respiration slow and deep. Unconscious. Arm and leg, left side, rigid. 15th.—Pulse rapid and small; temp. 101°. 16th.—Con-vulsions. Death	16 days.	suture not congested. Con-volutions not flattened, slightly sticky. Slight ex-cess of fluid in lateral ventri-cles. Pia mater over optic commissure opaque; many scattered tubercles; little inflammatory change. Tu-bercles also in lungs and liver	16 days.	Coronal suture marked exter-nally by a red line. Little blood in superior longi-tudinal sinus. Convolutions flattened and sticky. Much fluid in lateral ventricles and subarachnoid space. Choroid plexus and sub-stance of brain rather pale. Pia mater over optic com-missure natural. Many tu-bercles scattered about; little lymph. Caseous bron-chial glands. Tubercles in lungs, liver, spleen, and kid-neys	16 days.

Name. Age. Remarks.	History of case before ophthalmoscopic examination.	Date of examination from onset and condition of fundus of eyes.	Symptoms present when the optic disks were first examined, and subsequent course of the disease.	Post mortem.	Duration.
<p>Case 6. Emily S., 2½ years. Dr. Dickinson.</p> <p>son. Marked rigidity. Evidence of intracranial pressure</p>	<p>Cough and wasting for six weeks. Sick twice a fort- night ago. Doubt- ful headache, worse for the last five days</p>	<p>14th day.—Left disk a trifle reddened. Right veins dark and enlarged; disk pale. 16th.—Left disk pale; margin lost, except outside. Veins large and dark; arte- ries small. Right same as left, but vessels hidden by exudation at the margin of the disk</p>	<p>14th day.—Quite unconscious; weakening of left facial. Pulse 148, regular. 15th.— Eyes not parallel; groaning; legs and right elbow rigid. 16th.—Flushed; convulsed in right arm. 18th.—Quite unconscious. Pulse 80, most irregular. 19th.—Deep scarlet rash on both arms; rigidity of both arms and legs; nystagmus. Tempera- ture rose till death to 107°</p>	<p>Coronal suture natural. Super- rior longitudinal sinus a small clot. Convulsions flat and sticky. Excess of ventricular fluid. Lymph about optic commissure. Optic nerves not reddened. Brain substance softened around the lateral ventricles. Many tubercles. Caseous bronchial glands. Tubercles in lungs, liver, spleen, and kidneys</p>	19 days.
<p>Case 7. Harry W., 3 years. Dr. Dickinson</p>	<p>Vomiting and head- ache from onset; restless nights; flushing of face; constipation. His- tory of congenital syphilis</p>	<p>9th day.—Both disks pale; margins quite lost; vessels obliterated at the margins. Veins large and arteries small. 10th. —Disks the same</p>	<p>9th day.—Flushed, semi-con- scious; external strabismus right eye; palsy left face, arm, and leg. 10th.—Pulse 78, irregular; unconscious. 11th.—Deeply comatose; pulse 100, regular; convulsed face, arms, and legs. 12th. —Respirations irregular; pulse 160 at least; rigidity both arms and legs. Death 6th day.—Sick this morning, and complaining of headache; tongue furred. Pulse 80, sub- regular. Not looking ill; reading a book. 7th.—Pulse 72. Palsy left side, face, arm, and leg; external squint left eye. 8th.—Still vomiting. Pulse 78, irregular. 9th.—</p>	<p>Pia mater sticky; excess of ventricular fluid; choroid plexus pale; lymph and tu- bercles round chiasma. Some opacity of retina round optic papilla. Caseous bronchial glands; tubercles in lungs, spleen, and liver</p>	12 days.
<p>Case 8. John M., 9 years. Dr. Dickinson.</p> <p>Diagnosis aided by the ophthalmos- cope</p>	<p>Bronchitis six weeks ago. Lately occi- pital headache; restless nights; bowels confined. Not a strong child</p>	<p>6th day.—Both disks pale; margins obscured, except outer side of right; veins hazy; no great disproportion be- tween arteries and veins. 9th.—Veins larger; some swelling; a white parallel band each side</p>	<p>Convulsions sticky; much subarachnoid fluid; if ven- tricular excess. Choroid plexus pale; optic nerve swollen just behind the eye- ball; optic chiasma red and soft; brain substance gene- rally soft; tubercles in the membranes, numerous and</p>		12 days.

Case 9. William H., 3 years. Mr. Smith. Vessel changes chiefly. Marked and early rigidity. Large sub- arachnoid excess	1st day.—Vomiting. 5th.—Screaming. 12th.—Vomiting; flushing; right in- ternal squint; re- tracted head. A case of old hip dis- ease	of the arteries in right. 12th.—Disks not so pale	Speech thick; abdomen re- tracted. 10th.—Drowsy. Pulse 96. Palsy less marked. 11th.—Pulse 124, regular. 12th.—Convulsed both sides; insensible; arms and legs rigid. Pulse 160; respiration 64	fine. Caseous bronchial glands; tubercles in liver, spleen, and kidneys
	12th day.—Vomiting. change. 16th.—Right, vessel changes only, and these marked; veins twice the size of the arteries, which are very pale and small. 17th.— Both disks pale; same vessel changes in left; veins almost three times the size of arteries. 21st.—Much the same; vessel changes out of proportion to the disk	12th day.—Disks, no change. 16th.—Right, vessel changes only, and these marked; veins twice the size of the arteries, which are very pale and small. 17th.— Both disks pale; same vessel changes in left; veins almost three times the size of arteries. 21st.—Much the same; vessel changes out of proportion to the disk	12th day.—Typhic fever. Tem- perature 99° to 102°. Vomit- ing; tremor of head and right arm; internal squint of right eye; bowels confined. 15th.—Much headache, moaning, arms rigid. 16th. —Respiration 80, irregular. Convulsed right arm and side of face; unconscious. 18th. —Moaning, more conscious, flushing. Pulse frequent. 19th.—Convulsed in left hand. rigid; head turned to left. Pulse 11th day.—Pulse 104, intermit- tent. Sick once; furred tongue; headache; delirium; phos- phatic urine. 13th.—Ten- perature 100.4°. Otorrhoea. Pulse 140. Brown, dry tongue; right facial weak- ened. 14th.—Barely con- scious. Pulse irregular, 86. Screaming. 15th.—Right external squint. 16th.— More conscious. Pulse 88. Abdomen retracted. 18th.— Flushed face. Respiration irregular. Arms and legs rigid. Pulse 144. Head to left. Temperature 108.4° five minutes before death	Sutures not congested. Con- volutions not flattened or sticky; no ventricular excess of fluid, but much subarach- noid. Reddening of pia mater over optic commissure; little inflammatory exuda- tion; tubercles in meninges. Left optic disk looks natural to the naked eye. Bronchial glands caseous; tubercles in lungs and liver
Case 10. Robert M., 5 years. Dr. Dickinson. Membranes probably in- fected by tubercular masses in the brain. Optic changes most marked on the same side as the palsy	1st day.—Constant headache. 6th.— Vomiting since, daily; squint for three weeks; slight cough; bowels con- fined. Occasional headache for nine months	11th day.—Disks natural. 14th.—Right disk pale; obscured margin, except outside; a white line each side of and parallel to the arteries. Vessel changes not great. Left, same changes, but rather less; vessels hidden near the margin from exuda- tion. 15th.—Right, more exudation. 17th. —Changes advanced in right, not in left. 18th. —Vessels of right dip- ping near the margin; veins enlarged in both disks, and arteries small	20th.—Unconscious. 21st.—Arms rigid. Pulse 160. Death A red line along coronal suture. Convulsions flat and sticky. Superior longitudinal sinus small. Large excess of fluid in ventricles and subarach- noid. Chiasma reddened and embedded in thickened pia mater. Optic nerves red- dened; sheath distended just behind eyeball. A tubercu- lar mass, size of small bean, on upper surface of cerebel- lum; another, smaller, in left optic thalamus. Substance of brain soft. Many tuber- cles in membranes. Margins of disks to the naked eye obscured as during life. No tubercles in other organs	

Name. Age. Remarks.	History of case before ophthalmoscopic examination.	Date of examination from onset and condition of fundus of eyes.	Symptoms present when the optic disks were first examined, and subsequent course of the disease.	Post mortem.	Duration.
Case 11. Thomas M., 24 years. Dr. Williams. Chorea in the first year of life; con- vulsions; in- termittent fever; and marked facial weakness.	1st day.—Vomiting; cross; bowels constipated; restless night. 4th. — Squinting; abdomen flattened; delirium; respira- tion irregular; left facial weakened	5th day.—Left disk, veins enlarged and small, proportion as two to one. No other change. Right, veins not quite so large. 7th.—Right, the same. Left, not visible from condition of cornea	5th day.—Unconscious. Pulse 152, regular. Abdomen flat. Lies on left side. 7th.— Head retracted; left hand rarely moved; left cornea excoriated. Pulse 156. Un- conscious. 8th.—Tremor hands and legs. No distinct unilateral palsy. 9th.—Death	A red line along coronal suture. Convulsions flat and sticky. Much fluid in lateral ventri- cles; little subarachnoid. Pia mater over chiasma thick- ened. No swelling of optic sheath. Disks well defined	9 days.
Case 12. Perry H., 7 years. Dr. Williams. First illness at age of one year; marked facial weakness.	Vomiting, headache, and prostration for several days. Di- lating pupils for three days. Marked facial weakness.	14th day.—Right disk, veins dark; no other change. Left pale and uneven in colouring; margin quite obscured; slight swelling. Veins dark and enlarged. Arteries small. 17th.— Right, as before. Left, vessels obscured at mar- gins of disk, and more changed	14th day.—Flushed; sensible. Pulse 104, regular. Di- plopia with left internal strabismus. 15th.—Drowsy, but not looking ill. Urine phosphatic. Pulse 102, regular. 17th.—Pulse 102. Unconscious. Abdomen flat- tened. Right facial weak- ened, and twitching in right arm. 18th.—Death	Ventricles much dilated. Tu- bercles on chiasma. Soft- ening of brain. Many tubercles in Sylvian fissures, especially right. Tubercles in lungs, liver, and spleen.	18 days.
Case 13. Dr. Williams. Chorea in the first year of life; con- vulsions; intermittent fever; and marked facial weakness.	Marked facial weakness.	14th day.—Left disk red- dened, and margin ob- scured, except at outer one third. Veins dark, barely enlarged. 19th. —Left disk veins deci- dedly enlarged and dark. Same condition in right vessels, obscured at the margin. 21st.—Disks much the same; vessels obscured beyond the margin	14th day.—Pulse regular. Irritable. Left facial weak- ened. 19th.—Retching. Half unconscious. 20th.— Unconscious; flushed. Pulse 140, regular. Cheyne Stokes's respiration. 21st. —Respiration 102; pulse 172. No palsy; twitching of right side. 22nd.—Con- vulsed all over. Death	Convulsions flattened; excess of ventricular fluid and of subarachnoid. Pia mater contains many tubercles; little inflammatory change. Chiasma red. Choroid plexus pale. Brain substance soft- ened. Caseous bronchial glands. Tubercles in lungs, liver, spleen, and kidneys	22 days.

<p>Case 15. Beatrice S., 3 years. Dr. Chesdole, No changes in disks</p>	<p>For two months a slight cough. For nine days paroxysmal cough, ending sometimes in vomit- ing. Tongue fur- red. Pulse regular. Slight signs at apex of left lung. Com- plained once of headache</p>	<p>10th day.—Right disk, general reddening; faint margin, inner half only. Veins dark, twice the size of the arteries. 11th.—Left disk just the same as right, but slight swelling and ex- udation along the ves- sels. 14th.—Right disk paler. 15th.—Left paler in the centre; margin quite lost; some swelling</p>	<p>10th day.—Pulse 100. Sick a few times; flushed; bowels costive. Nothing obviously cerebral in the appearance of the disease. 11th.—Pulse 90, regular. A bad night; now more drowsy. Right facial weakened. 13th.— Respirations irregular; un- conscious; some twitching; abdomen flattened. 14th.— Pulse 160. Right facial as before. 15th.—Pulse 148, small. Convulsed both sides. Death</p>	<p>15 days. Coronal suture somewhat red- dened. Convulsions flat- tened. Excess of ventri- cular fluid, and probably of subarachnoid. Pia mater over chiasma thickened. Scattered tubercles. Brain substance softened round the ventricles. Bronchial glands caseous. Tubercles in the liver and lungs</p>	<p>Case 16. Amelia D., 13 years. Dr. Dickinson. A rather chronic lung case, with optic changes ill marked</p>	<p>Dull, heavy, and fall- ing away the last month. Sometimes quite cheerful. Occasional com- plaint of headache. For the last fort- night sick daily after food. Bowels confined</p>	<p>15th day.—No change in the disks. 17th.— Slight marginal red- dening of left disk. Right natural. 18th. —Much the same</p>	<p>15th day.—Irritable. Pulse 68, irregular. Slight chest signs; no headache. 16th.— Drowsy. Right facial weak- ened. 17th.—Unconscious. Right arm rigid. 18th.— Internal strabismus of left eye. Nystagmus. 19th.— Still unconscious. Both arms rigid. Pulse 148; temp. 101.6°. Death</p>	<p>19 days. Convulsions flattened and sticky. Much subarachnoid fluid. Optic nerve pale. Tubercles numerous on left hemisphere. Caseous bron- chial glands. Lungs con- tain many tubercles; also liver and spleen</p>	<p>21 days. Superior longitudinal sinus natural. Convulsions flat- tened and a little sticky. Ventricles distended. Much lymph at base and tubercles. Optic nerves natural. No softening of brain substance. Collapse and catarrhal pneu- monia of lungs. Caseous bronchial glands. Tuber- cles in liver, spleen, kidneys, peritoneum, and pericardium</p>
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Name. Age. Remarks.	History of case before ophthalmoscopic examination.	Date of examination from onset and condition of fundus of eyes.	Symptoms present when the optic disks were first examined, and subsequent course of the disease.	Post mortem.	Duration.
<p>Case 17. Adolphus M., 4½ years. Dr. Cheedle. Changes mainly in the retinal vessels. Much intra- cranial pres- sure; little inflammation. Diagnosis aided by the ophthalmo- scope. Death from collapse of lung</p>	<p>1st day.—Vomiting and pains all over. 6th. — Vomiting; headache; bowels confined. Wasting a little for the last three months. The father and two other children died of phthisis</p>	<p>8th day.—Right disk, slight general reddening; margin clear all round; veins twice the size of the arteries. Left, more reddening; vessels as opposite; veins dark, a margin only outside</p>	<p>8th day.—Pulse 100, irregular. Headache; irritability; screaming; head retracted. Respirations 20. 10th.— Sudden dyspnoea. Respira- tions 72. Death</p>	<p>Sutures and superior longitu- dinal sinus natural. Convo- lutions flat and sticky. Excess of ventricular and subarach- noid fluid. Very slight red- dening and thickening of membranes over chiasma. A few tubercles and some lymph in left Sylvian fissure; other- wise little of either. A small tubercular mass in left hemi- sphere of cerebellum. Col- lapse of three quarters of left lung caused death</p>	<p>10 days.</p>
<p>Case 18. Florence D., 8 years. Dr. Gee. Death from lung disease. Improvement in cerebral symptoms and in optic disks before death</p>	<p>Caught cold five weeks ago; cough since and wasting; feverish. Bowels confined. Seven days ago commen- ced to vomit, and two days com- plained of headache. A delicate, pale child. Dry, hack- ling cough; some catarrh about the apices of the lung. Pulse regular.</p>	<p>10th day.—Slight general reddening of both disks, with obscuration of the margin except exten- sively; vessel changes in both, but slight. 18th. — Changes advanced both in vessels and disks; left most. 16th. — Disks paler; margins more blurred; veins large and dark; arteries very small. 19th.— Disks very pale, swollen; vessels not hidden. 21st.</p>	<p>10th day.—Cough; paroxys- mal headache; furred tongue. 11th.—Pulse 144; flushing; vomiting. 12th.—Same sym- ptoms. Pulse 140; tempe- rature 100° to 102°. 18th.— Vomiting; diplopia; diver- gent squint; right facial weak. 14th.—Drowsy; in- coherent. 15th.— Mental blank. Pulse 144, regular. Veins at inner angle of eye full. 16th.—Abdomen re- tracted; more conscious. 19th.—Respirations irregu-</p>	<p>Convolutions not flat or sticky. Veins of pia mater on con- vexity full. Many tubercles and hyperamia of membranes about optic chiasma. Optic nerves barely reddened. Cho- roid plexus pale. Tubercles on velum interpositum. Sub- stance of brain not softened. No excess of ventricular fluid. Bronchial glands caseous. Both lungs crammed with tubercles; barely any breath- ing room. Liver, spleen, and kidneys tubercular</p>	<p>28 days.</p>

Three other children died of strumous complaints	—Disks not so pale. 22nd.—Right disk decidedly redder, more uneven in colouring; an indication of a margin outside. Left disk redder at lower and inner part	lar; some consciousness; right arm weakened. 22nd.—Still conscious; squint and palsy less. Cyanotic respiration, 48; pulse 144. Bowels loose. 23rd.—Unconscious only for nine hours before death	4 days.	Sutures congested. Convolutions flat. Chiasma and nerves reddened. Much sub-arachnoid fluid and some ventricular excess. Choroid plexus pale. Little lymph in pia mater, but tubercles. Sheath of optic nerve swollen. Caseous bronchial glands. Tubercles in liver and spleen	14 days.
Case 19. John M., 4 years. Dr. Dickinson. Changes chiefly in the vessels. Marked intracranial pressure	Imperfect history. 1st day.—Irritable; pale and prostrate. Temperature 100.6° to 101.4°. Restless at nights	3rd day.—Slight reddening of both disks, with marked disproportion in size between the arteries and veins, about 2 to 1. 4th.—Veins very dark and turgid	3rd day.—Lies on left side; eyes turned to left; head retracted; weakening of left facial. Pulse 100, regular. 4th.—Convulsions, both arms and legs. Death		
Case 20. Harriet S., Dr. Gee. Obscure symptoms. Tubercles of choroid. Whole duration of illness 14 days	1st day.—Feverish; sore throat; loss of appetite. 2nd.—Sickness and headache; bad cough. 9th.—Delirious at night and often in the day, with headache; no squint. The child's mother died of phthisis	14th day.—Optic disks natural. In the right, and internal to the optic nerve, a white, shining, defined patch, about a quarter the size of the disk, and crossed by a vessel; little pigment about it. Examined microscopically by Dr. Gowers, who found tubercles of the choroid	14th day.—Depressed and ill; sordes on lips; offensive breath and typhoid look; quite rational; no headache; abdomen full; a hard cough. Respiration 84, with only signs of bronchitis. Slight cyanosis. Active delirium that evening, and death at 1 a.m., preceded for two hours by convulsions	Sinuses and veins of pia mater about natural. No flattening of convolutions or stickiness. Moderate amount of lymph, and a few tubercles in right Sylvian fissure. No inflammation of membranes round the optic commissure or nerves. No excess of ventricular fluid; slight of sub-arachnoid. Some white spots in the choroids. Caseous bronchial glands. Tubercles in lungs, liver, spleen, and kidneys. Tubercular ulcers of the intestines	

Name. Age. Remarks.	History of case before ophthalmoscopic examination.	Date of examination from onset and condition of fundus of eyes.	Symptoms present when the optic disks were first examined, and subsequent course of the disease.	Post mortem.	Duration.
Case 21. Edward P., 6 years. Mr. Owen. Chronic hip- joint disease. Optic changes not great. Early rigidity. Intra-cranial pressure	1st day.—Change of temper, child be- coming irritable, inert, and drowsy. 4th. — Vomited. 5th.—Constant vo- miting. 7th.— Unconscious all day. Convulsed right side; bowels con- fined; abdomen retracted. Tempe- rature below 101° for the last two days; before this 101° to 108°. A case of hip-joint disease of eight months' standing; excision performed Wasting for the last two months; cough and yellow expecto- ration. Signs of cavity at left apex	8th day.—Veins of disk slightly enlarged; no other change. 9th.— Vessel changes distinct; arteries small and pale; veins large and rather dark. No other change; disks pale. 11th.— Same condition of disks	8th day.—Pulse 144, regular. Temperature 100°. Flushed face; divergent squint; both arms and right leg rigid; abdomen flat; no tache. 9th. —Pulse 160, full, soft, and regular. Rigidity and con- sciousness the same; swallows nothing. 10th.—Conscious, but does not speak; rigidity gone. Pulse 130. Swallow- ing nothing. 11th.—Still conscious. 12th. — Died, 2 a.m.	Slight compression of superior longitudinal sinus. Convo- lutions flat and sticky. Ex- cess of ventricular fluid; less of subarachnoid fluid. Tuber- cles both Sylvian fissures, but the merest trace of inflam- mation. No inflammation about optic nerve or com- missure, and no tubercles. Tubercles both lungs; liver albuminoid	12 days.
Case 22. Charlotte E., 6 years. Dr. Gee. Meningitis of left convexity. Palsy right side. No optic changes		1st day.—Optic disks perfectly natural. 16th. —Optic disks natural	1st day.—Sudden onset of convulsions, followed by palsy of right side, face, arm, and leg; sick once. 3rd.— No change; never speaks; conscious. 9th.—Pulse 120, regular. No sickness; bowels moved daily; febrile. 16th. —Febrile still; taking less notice; palsy less marked in leg. 19th.—Slight rigidity right elbow; still conscious. Died 10 p.m.	Base of brain free from tuber- cles. No ventricular excess; decided excess in subarach- noid. Many tubercles in con- vexity of brain, especially at bifurcation of left Sylvian fissure. Old cavity left lung and tubercles. Tubercles in liver, spleen, and kidneys	19 days.

<p>Case 22. Nally J., 7½ years. Dr. Gee. Diagnosis aided by the ophthalmo- scope. Chief changes seen about the optic commissure</p>	<p>A short cough for last six weeks, often followed by vomit- ing. Child wasting rapidly. For the last fortnight frontal headache and dislike of light. Nights restless. Drowsy. Never a strong child</p>	<p>14th day.—Right disk, a faint margin outside, the rest lost. Grey-red colour and uneven. Some arteries obscured. Slight swelling. Veins dark and enlarged. Left much the same as right; more dispropor- tion between the arte- ries and veins. 18th.— Rather more edema of left disk. No other change. Owing to the child's extreme irrita- bility it was quite im- possible to again ex- amine the eyes</p>	<p>14th day.—No headache; drowsy. Pulse 104, regular; temp. 100.1° F. Tongue a thin fur. A short loose cough, with bronchitic signs in chest, chiefly at apices. No cerebral symptoms and no palsy. A fairly-marked tache. 15th.—Rather more bronchitis. Pulse quite regular. Noted by Dr. Gee. The notion of meningitis rested wholly upon the optic neuritis. 16th.—Restless night. No headache or vomiting. Pulse 128, regu- lar; temp. 101° to 102.4°. Nothing more definitely cerebral. 18th.—General tenderness and irritability. Complained, when asked, of headache. Pulse 140, regu- lar. No squint or other palsy. Abdomen flat. Bowels confined. 19th.— Restless night; screaming; now half comatose. Pulse regular. Occasional diver- gent squint. 24th.—Since last note drowsiness and great irritability. 25th.— More drowsy. Right facial weakened. Tongue brown; sordes. Abdomen blown up. 26th.—Palsy more marked. Unconscious a few hours before death</p>	<p>26 days. No congestion about sutures. Medium amount of blood in superior longitudinal sinus. Veins of pia mater con- tain much blood. Convolu- tions flat and sticky. Marked excess of ventricular fluid; some subarachnoid excess. Not much lymph in Sylvian fissures; a trifle more in right. Optic commissure embedded in lymph; tuber- cles with the lymph. Brain substance barely softened. Bronchial glands caseous. Tubercles in lungs, liver, spleen, and kidneys</p>
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Name. Age. Remarks.	History of case before ophthalmoscopic examination.	Date of examination from onset and condition of fundus of eyes.	Symptoms present when the optic disks were first examined, and subsequent course of the disease.	Post mortem.	Duration.
<p>Case 24. Stanley W., 6½ years. Dr. Cheadle. Changes in the disk and retinal vessels of right side. Palsy the same side. Chief brain affection on left side</p>	<p>Appetite falling off late. For two or three weeks com- plaining of any- thing glittering. 1st day.—Frontal headache. 4th.— Vomiting. Bowels confined. 10th.— Constant vomiting. Pulse 72, regular. Irritable and drowsy</p>	<p>15th day.—Right disk much reddened, espe- cially peripherally. Margin obscured all round. Slight swelling. Veins large and dark. Arteries small. Left quite normal. 18th.— Same condition of disks</p>	<p>15th day.—Impaired con- sciousness. Pulse 80, sub- regular; respiration irre- gular. Right facial and right arm weakened. Occa- sional scream. 16th.— Pulse 100, regular. More conscious. 18th.—Head re- tracted. 21st.—More un- conscious. Pulse 104, regu- lar. Bowels confined. Picks at left nostril. 22nd.— Pulse 168. Livid flush on cheeks. Deeply unconscious. Irregular respiration. 23rd. —Convulsed down the left side. Died at 10 a.m.</p>	<p>Pia mater sticky. Convul- sions flattened. Excess of fluid in lateral ventricles. Much lymph at the base of the brain, and scattered tubercles. Tubercles and inflammation in both Sylvian fissures, reach quite to the top in the left. A few tubercles in left lung</p>	<p>23 days.</p>
<p>Case 25. Thomas M., 4 years. Dr. Cheadle. Right disk most affected; right hemi- plegia. Men- inges of left side have most lymph. Changes in respiratory mechanism</p>	<p>A weakly child. Diarrhoea six weeks ago. 1st day.— Constant vomiting and drowsiness. 4th.—Vomiting still. Bowels con- fined. A fit</p>	<p>6th day.—Right disk inclined to striation; rather pale. Margin lost except a short dis- tance; vessel changes slight. Left, little change. 8th.—Left, rather more of upper margin blurred. 9th. —Vessel changes more pronounced. 12th.— Right disk swelling. Vessel changes marked. Arteries very small. Left somewhat ad- vanced</p>	<p>6th day.—Drowsy; sighing. Pulse 76, irregular. Furred tongue. Retracted abdo- men. Tache marked. 7th. —Right hemiplegia; squin- ting; more drowsy. Pulse 68, irregular. 8th.—Respi- ration mainly thoracic. Abdomen barely protrudes on inspiration. Pulse 84, most irregular; temp. 99°6'. Unconscious. 9th.—Rather more conscious. Pulse 100; respiration most irregular. 10th.—Picking at clothes. Quite unconscious. Respira-</p>	<p>Superior longitudinal sinus natural. Pia mater sticky; convulsions flattened. Lymph mounting left Syl- vian fissure. Pia mater over chiasma and tract thickened. Numerous tubercles. Turbid fluid in subarachnoid space, and swelling of sheath of optic nerve. Margin of right disk blurred; of left visible. Brain not cut into. Tubercles in the liver, and some in spleen cretaceous</p>	<p>12 days.</p>

<p>case 26. William C., 8 years. Dr. Dickinson. Diagnosis aided by the disk changes. Chief affec- tion of the meninges around the optic com- missure. Rigidity of limbs and great ventri- cular effusion</p>	<p>Two other children had harelip. This child had hooping- cough six months ago; coughing since, and occa- sional headache. 1st day.—Vomiting; wasting; bowels costive; drowsy. 8th.—Vomiting daily; feverish; clean tongue</p>	<p>9th day.—Right disk, slight reddening. Mar- gin lost except for a short distance outside. Veins large, and arte- ries small. Left redder; vessel and disk changes less than right. 16th. —Left disk changes advanced in vessels and disk. Right as before. 16th.—Left disk paler; barely any margin. Veins twice the size of arteries. Right, less margin. 18th.—Left, disk arteries partly hidden from exundation. Right much as left</p>	<p>tion wholly abdominal. Ab- domen concave on expiration. Unconscious. 11th.—Pulse 132, regular. Swallows badly. Head retracted. 12th.—Pulse 180; respira- tion wholly thoracic. Death</p>	<p>20 days.</p>
			<p>Slight line of congestion along the sutures. Superior longi- tudinal sinus smallish. Con- volutions much flattened and sticky. Great excess of ven- tricular fluid; little sub- arachnoid. Sheath of optic nerve barely any fluid. Right Sylvian fissure a few tubercles and lymph. Basal meningitis expended around optic commissure, and nerves here thick with lymph. Optic nerves swollen; many vascular points. Veins of Galen empty. Brain sub- stance softened. Disks show obliteration of margins. Caseous tracheal glands. Tubercles in lungs, liver, spleen, kidneys, and peri- toneum</p>	

INDEX.

These Indices to the annual volumes are made on the same principle as, and are in continuation of, the General Index to the first fifty-three volumes of the 'Transactions.' They are inserted, as soon as printed, in the Library copy, where the entire Index to the current date may always be consulted.

- ABSCCESS of the brain, traumatic, case of secondary trephining for; recovery (John W. Hulke) . . . 367
- ALBUMEN in the urine in croup, replies relative to occurrence of . . . 48
- in chronic nephritis, tables showing amount of (E. I. Sparks and J. M. Bruce) . . . 258-68
- ALBUMINURIA, comparative presence of, in cases of membranous croup and diphtheria . . . 18, 26
- ANEURISM, AOERTIC, on deligation of the right carotid and subclavian arteries for, with a new species of ligature (Rd. Barwell) . . . 393
- History of the case, 393; the operation chosen, 395; ligature used—objections to those hitherto used—silk, twine, catgut, 401-3; experiments in making a flat tape-like ligature from the aorta of the ox, and removing its superfluous elasticity, 404-5.
- ANEURISM involving the innominate artery, on three cases of distal ligature of the carotid and subclavian arteries (Rd. Barwell) . . . 217
- Reference to previous case, 'Trans.,' vol. lxi, pp. 13, 217; history and progress of Cases 2 and 3, 218-24; Case 4, 224-28; Remarks, organisable ligatures, 229-30.
- ANKLE clonus phenomena (W. R. Gowers) . . . 283-304
- see *Tendon-reflex*.
- ARTERIES, CAROTID and SUBCLAVIAN, distal ligature of, for aneurism involving the innominate artery, three cases (Rd. Barwell) . . . 217
- RIGHT CAROTID and SUBCLAVIAN, deligation of, for aortic aneurism, with a new species of ligature (Rd. Barwell) 393
- VOL. LXII. 31

ARTERY, INNOMINATE, see *Aneurism of.*

- SUBCLAVIAN, deligation of, for aortic aneurism (Rd. Barwell). 393

BARWELL, Richard.

On three cases of distal ligature of the carotid and subclavian arteries for aneurisms involving the innominate artery 217

On deligation, for aortic aneurism, of the right carotid and subclavian arteries, with a new species of ligature 393

BLADDER, RUPTURED, on the diagnosis and treatment of (Christ. Heath) 335

Detailed report and post-mortem of the case by Samuel Burton, Surg.-Registrar Univ. Coll. Hospital, 336-40; references to other cases by Mr. Willett, Mr. Chaldecott, Dr. Walter, Dr. Henley Thorp, and Dr. Mason, 341-45.

- tumour in, removed by perineal incision; complete recovery (G. M. Humphry) 421

Only other similar case, one in the practice of Prof. Billroth, 423; summary of the characters of growths in the bladder, 424-26.

BRAIN, traumatic abscess of, case of secondary trephining for, recovery (John W. Hulke) 367**BRETONNEAU's Memoirs on diphtheria, notice of 72-4****BRUCE, J. Mitchell, M.D., see *Sparks* and Bruce on chronic nephritis.****BURTON, Samuel**

Details of report, Mr. Christopher Heath's case of ruptured bladder 336

BUTLIN, Henry T., see *Savory* and Butlin, perforating ulcer of the foot.**CATARACT, remarks on one hundred and fifty operations for extraction of (Charles Higgens) 347**

Table of the cases of operations, 355-65.

CELLS, GIANT, appearances so described, in the pathology of lupus (Geo. Thin) 407**CHALDECOTT, —.**

Case of ruptured bladder, referred to (C. Heath) 342

CHEYNE, John

On "cynanche trachealis, or croup," 1801, notice 70

CLONUS, see *Ankle Clonus.*

COLD, origin of membranous laryngitis from definite exposure to . . . 15

COMMITTEE (SCIENTIFIC) OF THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY.

REPORT on the relations of MEMBRANOUS CROUP and DIPHTHERIA. [Summary of conclusions, p. 31] . . . 1

[W. Howship Dickinson, *Chairman*, C. Hilton Fagge, M.D., Samuel Gee, M.D., J. F. Payne, M.D., H. G. Howse, B. H. Semple, M.D., and W. S. Greenfield, *Secretary*.]

Object and scope of the inquiry, 3. I. Causation, 6. II. Conditions of occurrence: season, weather, climate, 10; hygienic condition, 11; association with other diseases, 13; origin of membranous laryngitis from a definite exposure to cold, 15. III. Course and symptoms in individual cases, mode of onset, &c., 15-20; relations of membranous and non-membranous laryngitis, 20; definition of the word croup, 27; definition of the word diphtheria, 30; summary of conclusions, 31; copies of circulars of inquiries, 34-7. APPENDICES: I. Digest of replies to queries, 39. II. Report on the history and early literature of the subject, 67. III. Historical sketch of anatomical distinctions which have been drawn between croup and diphtheria, 77; history of the anatomy of diphtheria in England, 77; ditto in Germany, 80. IV. Report on the histology of the laryngeal and tracheal false membrane, 89; case of membranous laryngitis from Eau de Cologne, by Dr. Whitehead Reid, 95. V. Dr. Dickinson's Tables of Cases, with remarks, 101. VI. Dr. Hilton Fagge's Collection of Cases, 127. VII. Dr. Gee's Tables of Cases, 149. VIII. List of Reports to the Medical Officers of the Privy Council and Local Government Board on outbreaks of Diphtheria and subjects relating thereto, 167.

CROUP, evidence as to its relation with other diseases . . . 46-8

CROUP, MEMBRANOUS, and DIPHTHERIA, REPORT of the Committee on the relations of. [For details, see under *Committee*] . . . 1

CROUP and DIPHTHERIA, the relations of—

- history and early literature of the subject . . . 67
- history of anatomical distinctions drawn between them, 77
- scheme for the analysis of records of cases of existence of false membrane . . . 86-7

DALBY, W. B., M.B.

On disease of the mastoid bone [three cases] . . . 233

DELIGATION of right carotid and subclavian arteries, see *Arteries*.

DICKINSON, William Howship, M.D.

Tables of cases, with observations (Appendix V to the Report on Croup and Diphtheria) . . . 101-26

See *Committee* on Croup and Diphtheria (Report).

- DIET, &c., observations on the effect of, in chronic nephritis**
(E. I. Sparks and J. M. Bruce) 243
- DIPHTHERIA, definition of the word** 80
- history of the anatomy of, in England 77
 - ditto, in Germany 80
 - List of Reports to the Medical Officers of the Privy Council and Local Government Board on outbreaks of (Appendix VIII to the Report on Croup and Diphtheria) 167
 - evidence as to its association with tonsillitis and other diseases 42-5
 - and MEMBRANOUS CROUP, REPORT of the Committee on the relations of [for details see under *Committee*] 1
- EXERCISE, observations on the effect of, in chronic nephritis**
(E. I. Sparks and J. M. Bruce) 243, 256
- EYES, partial symmetrical immobility of, with ptosis (ophthalmoplegia externa) (Jon. Hutchinson)** 307
- see *Ophthalmoscopic* appearances in tubercular meningitis.
- EYELIDS, the movements of the (W. B. Gowers)** 429
- Explanation of the mechanism of the normal movements, 429-30; movements of the lower eyelid, 431; of the upper eyelid, 433; group of the associations of muscles in looking down and closing the eyelids, 436; case of paralysis of third nerve, 437; objection to Sir C. Bell's opinion on the effects of the contraction of the levator palpebræ superioris, 439
- FAGGE, Hilton, M.D.**
- Collection of cases (Appendix VI to the Report on Croup and Diphtheria) 127-48
 - See *Committee on Croup and Diphtheria (Report)*.
- FOOT, perforating ulcer of, cases, with remarks and an appendix on the literature of the subject (Wm. S. Savory and H. T. Butlin)** 373
- GARLICK, George, M.D.**
- Observations on the ophthalmoscopic appearances in the tubercular meningitis of children 441
- GASKOIN, George.**
- Case of Morphaea 169
- GEE, Samuel, M.D.**
- Tables of cases (Appendix VII to the Report on Croup and Diphtheria) 149-65
 - See *Committee on Croup and Diphtheria (Report)*.

- GIANT CELLS, appearances so described in the pathology of
lupus (Geo. Thin) 407
- GOWERS, *William Richard*, M.D.
Study of the so-called tendon-reflex phenomena 269
The movements of the eyelids 429
- GOWERS, *William Richard*, M.D.
Microscopical examination of the brain in a case of ophthal-
moplegia externa (Jon. Hutchinson) 316
- HEATH, *Christopher*.
On the diagnosis and treatment of ruptured bladder 335
- HIGGINS, *Charles*.
Remarks on one hundred and fifty operations for extraction
of cataract [with the table of cases] 347
- HOLMES, T.
On the operation of tracheotomy, referred to 200
- HOME, F.
Description of "croup" in the east coast of Scotland, 1765,
notice 70
- HOWSE, *H. G.*, see *Committee on Croup and Diphtheria* (Report).
- HULKE, *John Whitaker*.
Case of secondary trephining for traumatic abscess of the
brain; recovery 367
- HUMPHRY, *George Murray*, M.D.
Tumour in the bladder removed by perineal incision; com-
plete recovery 421
- HUTCHINSON, *Jonathan*.
On ophthalmoplegia externa, or symmetrical immobility
(partial) of the eyes, with ptosis 307
Narrative of a case of true leprosy, in which complete re-
covery has taken place 331
- IODIDE of POTASSIUM, see *Potassium*.
- JURINE, *Louis*.
Essay on croup, notice 71
- TENDON-REFLEX phenomena (W. R. Gowers) 270-82
— see *Tendon-reflex*.
- TRACHEAL FALSE MEMBRANE, report on the histology of 89

- LARYNGITIS**, relations of membranous and non-membranous 20
- **MEMBRANOUS**, origin of, from a definite exposure to cold 15
 - — case of, from Eau de Cologne (Whitehead Reid) 95
 - — tracheotomy in, the indications for its adoption, and some special points as regards its after-treatment (Rt. W. Parker) 197
- LEPROSY**, narrative of a case of true, in which complete recovery has taken place (Jon. Hutchinson) . . . 331
- The leprosy acquired in Jamaica in 1835, 332.
- LETZNERICH**, Ludwig.
- On the pathology of diphtheria and croup, notice of . . . 85
- LIGATURE**, **DISTAL**, of the carotid and subclavian arteries for aneurism involving the innominate artery; three cases (Rd. Barwell) 217
- of right carotid and subclavian arteries, see *Arteries*.
- LIGATURES**, the value of organisable (R. Barwell) 229-30
- new species of [flat tape-like ligature from the aorta of the ox] (R. Barwell) 404-5
- LUPUS**, on the pathology of, with special reference to the appearances described as giant cells (Geo. Thin) . . 407
- Notice of previous writers on the pathology of lupus, 407-9; the lupus tissue from a case of Mr. Morrant Baker's at St. Bartholomew's Hospital, 410; description of the giant cells as made apparent by preparation in eosin dye, and their diagnosis, 412; question as to the cells being "disintegrated blood-corpuscles in degenerated blood-vessels," 413-15.
- MASON**, Erskine, M.D.
- Case of laceration of the bladder, recovery; referred to (C. Heath) 344
- MASTOID BONE**, on disease of the [three cases] (W. B. Dalby) 233
- Malignant disease excited by purely local irritation, and perforation of mastoid cells for averting the effects of the inflammation.
- MEMBRANES**, **FALSE**, laryngeal and tracheal; report on their histology 89
- MENINGITIS**, **TUBERCULAR**, of children, observations on the ophthalmoscopic appearances in (Geo. Garlick) . . 441
- Based on twenty-six cases at the Hospital for Sick Children, 441; summary of the changes observed, 442; questions as to their proportion and diagnostic value and condition of the brain membranes in relation to the optic changes, 444-7; table of cases in which the ophthalmoscope aided the diagnosis, 446; evidence of effective intracranial pressure in acute cases, 447-8; table of duration of the cases and affection of retinal vessels, &c., 449; post-mortem, 449; cases of recovery, 450-1; table of details of the cases, 452-63.

- MORPHŒA**, case of (Geo. Gaskoin) 169
- MUSCULAR SPASM**, see *Tendon-reflex*.
- NEPHRITIS, CHRONIC**, observations on the effect of diet, rest, exercise, &c., in (E. I. Sparks and J. M. Bruce) . . . 243
 Observations, series 1—6, ordinary milk and egg diets, 244-9; series 7, non-nitrogenous diet, 250; series 8, effect of rest, 251; series 9, experiments with digitalis, 253; general conclusions, 254; effects of non-nitrogenous diet, wine, and exercise, 255-6; effect of digitalis in excretion of albumen, 257; charts and tables showing the amount of albumen passed per hour in the series of observations, 258-68.
- OPHTHALMOPLÉGIA EXTERNA**, or symmetrical immobility (partial) of the eyes, with ptosis (Jon. Hutchinson) 307
 Reference to previous paper on ophthalmoplegia interna, 308; clinical histories of seventeen cases, 310-327; microscopical examination of the brain of Robert S., Case 3, by W. R. Gowers, M.D., 316; general comments on the series, 327.
- OPHTHALMOSCOPIC APPEARANCES** in the tubercular meningitis of children (Geo. Garlick) 441
- PARALYSIS** as a sequel of croup, replies relative to occurrence of 50
- PARKER, Robert William**.
 Tracheotomy in membranous laryngitis, the indications for its adoption, and some special points as regards its after-treatment 197
 — appendix of cases in which the after-treatment advocated in the paper was carried out 215
- PAYNE, J. F., M.D.**, see *Committee* on Croup and Diphtheria (Report).
- POTASSIUM, IODIDE OF**, nature of eruption from use of (Geo. Thin) 189
- PTOSIS** in cases of partial symmetrical immobility of the eyes (Jon. Hutchinson) 307
- REFLEX PHENOMENA**, see *Tendon-reflex phenomena*.
- REID, Whitehead**.
 Case of membranous laryngitis from Eau de Cologne . . . 95
- REPORT** of the Scientific Committee on the relation of membranous croup and diphtheria 1
 [For details, see under *Committee* (Scientific).]
- REST**, observations on the effect of, in chronic nephritis (E. I. Sparks and J. M. Bruce) 243, 251
- RUPTURE**, see *Bladder* (ruptured).

